A Dissertation on

A CLINICAL STUDY OF VARIOUS FINDINGS IN UPPER GASTROINTESTINAL ENDOSCOPY IN PATIENTS PRESENTING WITH LATE ONSET DYSPEPSIA

Dissertation submitted to

THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERISTY

CHENNAI

with partial fulfilment of the regulations

for the Award of the degree

M.S. (General Surgery)

Branch – I

MADRAS MEDICAL COLLEGE ,

CHENNAI.APRIL-2014

DECLARATION

I , certainly declare that this dissertation titled, "A CLINICAL STUDY OF VARIOUS FINDINGS IN UPPER GASTROINTESTINAL ENDOSCOPY IN PATIENTS PRESENTING WITH LATE ONSET DYSPEPSIA" ,represent a genuine work of mine . The contribution of any supervisors to the research are consistant with normal supervisory practice, and are acknowledged.

I , also affirm that this bonafide work or part of this work was not submitted by me or any others for any award , degree or diploma to any other university board , neither in India or abroad . This is submitted to The Tamil Nadu Dr.MGR Medical University, Chennai in partial fulfilment of the rules and regulation for the award of Master of Surgery Degree Branch 1 (General Surgery) .

Date :

Place : **Dr.S. SARAVANAKUMAR,**

BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of Dr.S.SARAVANAKUMAR on "A CLINICAL STUDY OF VARIOUS FINDING IN UPPER GASTROINTESTINAL ENDOSCOPY IN PATIENTS PRESENTING WITH LATE ONSET DYSPEPSIA" during his M.S. (General Surgery) course from may 2011 to April 2014 at the Madras Medical College and Government General Hospital, Chennai.

Prof.Dr.S.DEIVANAYAGAM, M.S.
Professor & Head of the Department,
Dept. of General Surgery,
Madras Medical College &
Government General Hospital,
Chennai – 600 003.

Prof.Dr.K.RAMASUBRAMANIAN, M.S.,
Professor of General Surgery,
Dept. of General Surgery,
Madras Medical College &
Government General Hospital,
Chennai – 600 003.

Prof.Dr.V.Kanagasabai M.D,

DEAN,

Madras Medical College & Government General Hospital, Chennai – 600 003.

ACKNOWLEDGEMENT

I would like to express my deep sense of gratitude to the Dean, Madras Medical College and also Professor and Head of the Department of General Surgery , MMC RGGGH, for allowing me to undertake this study on "A CLINICAL STUDY OF VARIOUS FINDING IN UPPER GASTROINTESTINAL ENDOSCOPY IN PATIENTS PRESENTING WITH LATE ONSET DYSPEPSIA". I was able to carry out my study to my fullest satisfaction, thanks to guidance, encouragement, motivation and constant supervision extended to me, by my beloved Chief Prof. Dr.RAMASUBRAMANIAN, Professor and Chief of General Surgical Unit. Hence my profuse thanks are due for him.

I am bound by ties of gratitude to my respected Assistant Professors, Dr.J.Antony Prabhakar , Dr.Umarani and Dr.Vijaya Lakshmi in general, for placing and guiding me on the right track from the very beginning of my career in Surgery and till this day. I would be failing in my duty if I don't place on record my sincere thanks to those patients who inspite of their sufferings extended their fullest co-operation.

I am fortunate to have my colleague PG's of our Unit Dr.jagadesan ,Dr.Murali,Dr.Gowtham,Dr.Arun,Dr.Inpharasun,Dr.H.Prasanna,Dr.GopiKrishnan,Dr.kesavan,Dr.kathiravan,Dr.iyyappa,Dr.ashok,Dr.anand,Dr.bharathi for their invaluable suggestions, relentless help for shouldering my responsibilities. Simply words cannot express its depth for their unseen contributions.

Lastly, my lovable thanks to my parents and brothers for their moral support

CONTENTS

ABSTRACT

Background and Objectives:

Uninvestigated dyspepsia is common in surgical out patient department. The prevalence of clinically significant upper gastrointestinal findings in late onset uninvestigated dyspepsia patients and their predictability based on history, is unknown. So a study was undertaken in Rajiv Gandhi Govt General Hospitals, Madras Medical College,Chennai to study the endoscopic findings in dyspeptic patients and to detect the esophagogastroduodenal carcinoma in early stages.

Materials and methods;

After informed consent 200 patients aged more than 40 years presenting with uninvestigated, untreated and uncomplicated dyspepsia were enrolled and evaluated in the study. Patients aged less than 40 years, patients on Proton pump inhibitors, patients who are known cases of chronic pancreatitis and liver disease, patients on NSAID's for more than one month duration, patients who had received Anti-Helicobacter pylori treatment and unwilling or unfit patients for endoscopy were excluded from the study.

All patients underwent upper gastro-intestinal endoscopy to document the various findings. Biopsies were taken in every patient from the gastric antrum and pathological site. The biopsy specimen was subjected to histo pathological examination for confirmation .The findings were documented and analysed.

RESULTS;

1. Highest prevalence of late onset dyspepsia in the age group of 41-50years

2. Most common presenting complaint was epigastric pain and discomfort

3. Dyspepsia was more common in males (59%) when compared to females

4. Most common endoscopic finding was gastritis followed by GERD

5. Malignancy was diagnosed in 6.5% patients with dyspepsia.

6. Clinically significant endoscopic findings were observed in 82.5% of patients with uninvestigated dyspepsia.

7. Most patients presented with a complex of three or more dyspeptic symptoms and the symptom profile was not predictive of the endoscopic findings. However, the high prevalence of gastritis (28%), suggests that most patients presenting with uninvestigated dyspepsia can be safely managed initially with acid suppressive drugs.

Conclusion:

Clinically significant endoscopic findings were observed in 82.5% of patients with uninvestigated dyspepsia. Most patients presented with a complex of three or more dyspeptic symptoms and the symptom profile was not predictive of the endoscopic findings. A larger number of inflammatory lesions as a result of increased acid production and low incidence of malignancy in the study group. It is suggested that the uninvestigated patients with dyspepsia may be initially managed medically with acid suppressive therapy .

Endoscopy may be undertaken in patients with recurrent symptoms or in whom drug therapy fails

Key words: Upper GI endoscopy; dyspepsia; H.pylori; GERD.

INTRODUCTION

Dyspepsia (also called uninvestigated dyspepsia) had been defined by the Rome working teams as pain or discomfort centred in the upper abdomen. Pain in the central portion of the abdomen is a key symptom, pain located in other areas or related to defecation is excluded. Discomfort is considered to be distinct from pain; however, both often coexist and the distinction may in part be culturally driven’’.

Discomfort has been defined as a subjective negative feeling that may include a variety of symptoms such as fullness in the upper abdomen, early satiety, bloating or nause.1,2

The definition of dyspepsia includes patients who have intermittent or continuous symptoms and does not specify the duration of symptoms. Thus dyspepsia may be of short or long duration, but acute self-limited dyspepsia does not usually require investigation and will not be considered further here.

The majority of patients who present with chronic dyspepsia have no obvious underlying explanation despite appropriate investigation; these cases are currently labelled as having non-ulcer (or functional) dyspepsia, although this is likely to be a heterogeneous condition.3 The pathophysiology of functional dyspepsia remains relatively poorly defined, but sensory and motor disorders of the stomach and duodenum appear to play a central role in at least a subset of cases.4

AIMS AND OBJECTIVES

To study of upper GI endoscopy findings in dyspeptic patient

1)The outcome of Upper GI endoscopy in dyspeptic patients

2) The co-relation of alarm symptoms with GI endoscopy finding

INCLUSION CRITERIA:

1) patient age above 40yrs with dyspeptic symptoms.

2) Patient with alarm symptoms.

3) Patient with previously diagnosed and treated cases of gastric ulcer, duodenal ulcer, complicated peptic ulcer, coming with dyspeptic symptoms.

EXCLUSION CRITERIA:

1) Patients who are endoscopy negative but have dyspepsia, may have motility disorders.

2) Individuals who had an endoscopy indications other than dyspepsia.

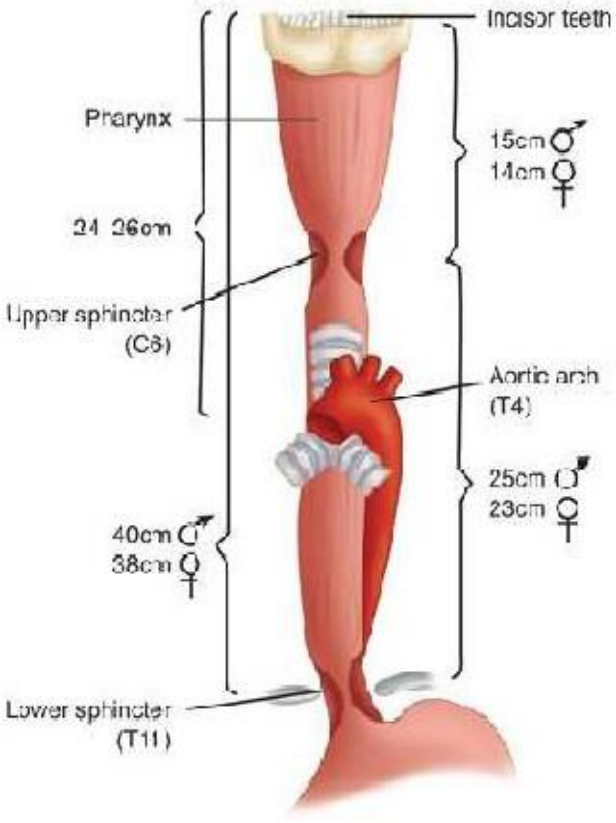
3) Not willing for endoscopy.

REVIEW OF LITERATURE

ANATOMY

ESOPHAGUS:

The esophagus, a soft muscular tube, allows food to pass between the pharynx and the stomach. It is about 25-30cm in length. The esophagus is a midline structure anterior to the spine and posterior to the trachea. From its origin at the cricoid cartilage in the neck opposite the fifth to sixth cervical vertebra, it passes into the thorax at the level of the sternal notch and travels caudally within the chest in the posterior mediastinum. It terminates in the abdomen at the esophagogastric junction opposite the twelfth thoracic vertebra. The esophageal hiatus of the diaphragm is at the level of the tenth thoracic vertebra.20



Anatomically esophagus is divided into three parts:

- Cervical
- Thoracic
- Abdominal

Function divides the esophagus according to its differing forms of motility into the following three zones According to differing forms of motility (functionally) esophagus is divided into three zones:

- Upper esophageal sphincter (UES)
- Esophageal body
- Lower esophageal sphincter (LES).

UPPER ESOPHAGEAL SPHINCTER (UES).

The high-pressure zone at the inlet of the esophagus is considered as UES. Anatomically it marks the end of a complex configuration of muscles that begin in the larynx and posterior pharynx and end in the neck. The pharyngeal constrictor muscles are three consecutive muscles that begin at the base of the palate and end at the crest of the esophagus. The superior and middle pharyngeal constrictor muscles, as well as the oblique, transverse, and posterior cricoarytenoid muscles, are immediately proximal to the UES and serve to

anchor the pharynx and the larynx to structures in the mouth and palate. These muscles also aid in deglutition and speech, but are not responsible for the high pressures noted in the UES. The inferior pharyngeal constrictor muscle is the final bridge between the pharyngeal and esophageal musculature.

ESOPHAGEAL LAYERS:

The esophagus is comprised of two proper layers: the mucosa and the muscularis propria. It is distinguished from the other layers of the alimentary tract by its lack of a serosa. The mucosa is the innermost layer and consists of squamous epithelium for most of its course. The distal 1 to 2 cm of esophageal mucosa transitions to cardiac mucosa or junctional columnar epithelium at a point known as the Z-line. Within the mucosa, there are four distinct layers

1. Epithelium

2. Basement membrane

3. Lamina propria, and

4. Muscularis mucosa.

HISTOLOGY:

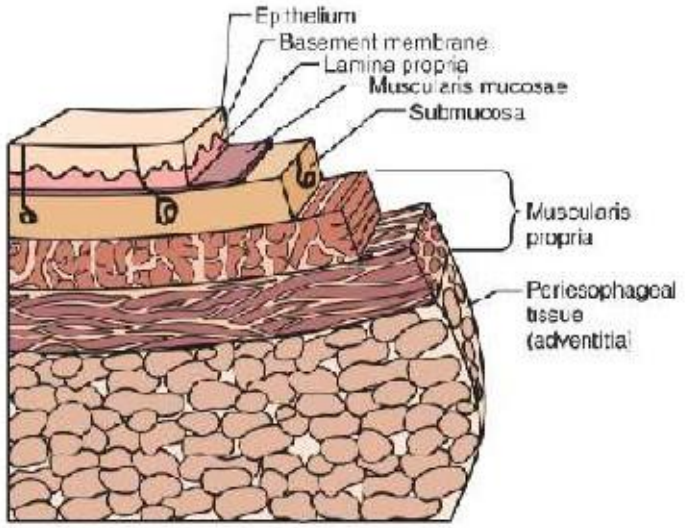


Fig 2. Histology of esophagus

upper third of the esophagus are striated, whereas the layers of the lower two thirds are smooth muscle. The circular muscles are an extension of the cricopharyngeus muscle and traverse through the thoracic cavity into the abdomen, where they become the middle circular muscles of the lesser curvature of the stomach. The collar of Helvetius marks the transition of the circular muscles of the esophagus to oblique muscles of the stomach at the incisura (cardiac notch). Between the layers of esophageal muscle is a thin septum comprising connective tissue, blood vessels and an interconnected network of ganglia known as Auerbach's plexus. Enshrouding the inner circular layer, the longitudinal muscles of the esophagus begin at the cricoid cartilage and extend into the abdomen, where they join the longitudinal musculature of

the cardia of the stomach. The esophagus is then wrapped by a layer of fibroalveolar adventitia.

ESOPHAGEAL CONSTRICTIONS:

The esophageal silhouette resembles an hourglass. There are three distinct areas of narrowing that contribute to its shape. Measuring 14 mm in diameter, the cricopharyngeus muscle is the narrowest point of the gastrointestinal tract and marks the superior-most portion of the hourglass-shaped esophagus. Occurring just below the carina, where the left main-stem bronchus and aorta abut the esophagus, the bronchoaortic constriction at the level of the 4th thoracic vertebra creates the center narrowing and measures 15 to 17 mm. Finally, the diaphragmatic constriction, measuring 16 to 19 mm, marks the inferior portion of the hourglass and occurs where the esophagus passes through the diaphragm. Between these three distinct areas of anatomic constriction are two areas of dilation known as the superior and inferior dilations. Within these areas, the esophagus resumes the normal diameter for an adult and measures about 2.5 cm.

LOWER ESOPHAGEAL SPHINCTER (LES):

The final phase of esophageal bolus transit occurs through the LES. Although this is not a true sphincter, there is a distinct high-pressure zone that measures 2 to 5 cm in length and generates a resting pressure of 6 to 26 mm Hg. The LES is located both in the chest and the abdomen. A minimum total length of 2 cm, with at least 1 cm of intra-abdominal length, is required for normal LES function. The transition from the intrathoracic to the intra-abdominal sphincter is noted on a manometric tracing and known as the respiratory inversion point (RIP). At this point, the pressure of the esophagus changes from negative to positive with inspiration and positive to negative with expiration.

Peristaltic contractions alone do not generate enough force to open up the LES. Vagal-mediated relaxation of the LES occurs 1.5 to 2.5 seconds after pharyngeal swallowing and lasts 4 to 6 seconds. This flawlessly timed relaxation is needed to allow efficient transport of a food bolus out of the esophagus and into the stomach. A post-relaxation contraction of the LES occurs after the peristaltic wave has passed through the esophagus, allowing the LES to return to its baseline pressure, re-establishing a barrier to reflux. 21

STOMACH:

Stomach is the most dilated part of the alimentary tract, extending from the cardiac end to the pyloric end. The stomach is sub-divided into;

1. Fundus

2. Body

3. Pyloric Antrum

4. Pyloric Canal

Fundus is the part which rises above the level of cardiac end of the stomach.Body is that portion situated between the fundus and the level of incisura angularis in the lesser curvature of the stomach.

The pyloric part is situated below the body and consists of:

1. Pyloric antrum

2. Pyloric canal

It is in the pyloric antrum where Helicobacter pylori is most frequently colonized.

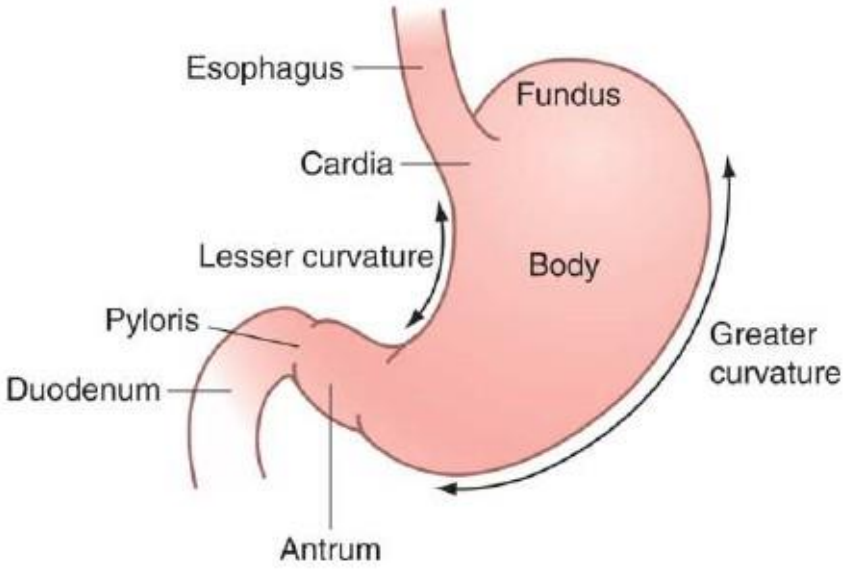


Fig 3. Anatomy of stomach

Stomach wall has four basic layers:

1. Mucous membrane

2. Sub mucosa

3. Muscular layer

4. Serosa

HISTOLOGY:

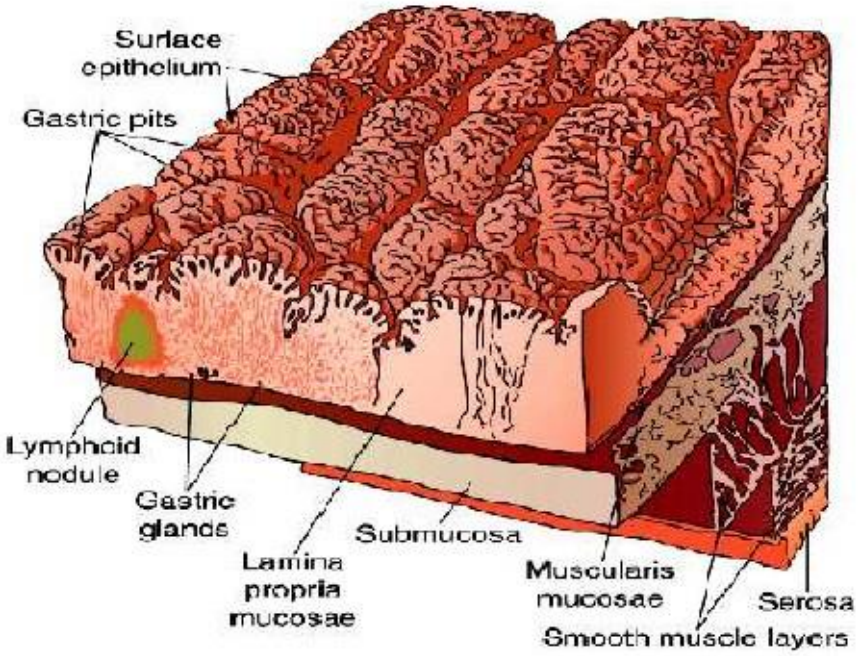


Fig 4. Histology of stomach

The Helicobacter pylori colonizes in the mucous layer of the gastric antrum and is important in relevance to its possible etiology of peptic ulcer disease.

Mucous membrane:

It is smooth and soft. To the naked eye, it appears as numerous folds (rugae) which disappear when stomach is distended. These rugae are most prominent towards the body and greater curvature and are less apparent in the antrum. The lining epithelium is a single layer of columnar cells which secrete mucus and are called“surface mucous cells”.

This surface epithelium dips into the lamina propria to form gastric pits. The mucosa is covered by a thick mucus layer, secreted from Surface mucous cells. The mucus acts as a lubricant and protects the stomach against its own acid and enzymes.

Damage to mucus layer exposes the stomach to gastric acid and active gastric enzymes and this is the basis of "Leaking Roof" hypothesis in the aetiology of peptic ulcers.22

Gastric glands are three types:

1) Cardiac glands

2) Main gastric glands

3) Pyloric glands

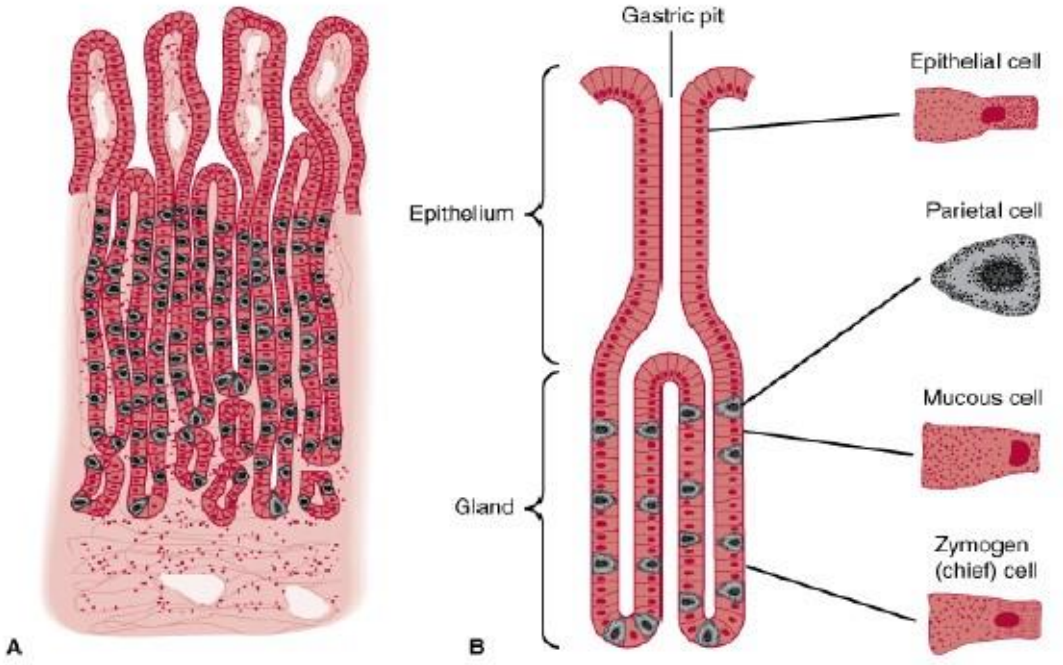


Fig 5. Different cells in gastric glands

1. Cardiac glands:

These are either simple tubular or tubulo-alveolar type confined to small area near the opening of oesophagus. They contain mainly mucus secreting cells.

2. Main gastric glands:

They are present in the fundus and body of the stomach and they open into gastric pits. They contain the following cells.

a. Chief cells: They are numerous in the basal parts of the glands. They secrete digestive enzymes like pepsin.

b. Parietal cells (Oxyntic cells): They are numerous in the upper part of the gland. They are responsible for the secretion of hydrochloric acid and intrinsic factor.

c. Mucous neck cells: They are present near the upper end of the gland and secrete mucous. Their secretions are different from that of the surface mucous cells.

d. Endocrine cells: These include somatostatin secreting D-cells and histamine secreting enterochromaffin-like cells. These are scattered throughout the glands.

e. Gastrin secreting cells (G-cells): Although small in number, they play a vital physiological role. They occur either singly or in small clusters in the mid to deep sections of antral glands. They contain basilar cytoplasm densely packed with gastrin containing secretory granules. The apical or luminal surface of the G-cells is narrowed into small microvilli, which are thought to contain the receptors responsible for the amino acid and peptide stimulation for gastrin release.

f. Undifferentiated cells: These are cells whose functions are not exactly known hence termed as undifferentiated cells.

3. Pyloric glands:

These are present in the antrum and pylorus. These extensively coiled glands are composed of endocrine, mucous and parietal cells. Mucous cells predominate in these glands.

PHYSIOLOGY:

The cells of gastric glands secrete about 2500ml of gastric juice daily. The juice contains mainly hydrochloric acid, pepsin, intrinsic factor, electrolytes and mucus.

Hydrochloric acid is produced by parietal cells in the body of stomach. It kills many ingested bacteria, provides the necessary pH for the pepsin to start protein digestion and stimulates the secretion of bile and pancreatic juice.23

The gastric mucosa is protected from the acid and pepsin by mucus content of gastric juice which forms a flexible gel that coats the mucosa. The surface mucous cells also secrete bicarbonate ions which are trapped in the mucus gel, so that the pH gradient which is established ranges from pH 1-2 at the luminal side, to pH 6-7 at the surface of the epithelial cells. The surface membrane of the mucosal cells and tight junctions between the cells are also part of the mucosal barrier, which prevents the back diffusion of H+ ions and protects the epithelium from damage.

Helicobacter pylori colonizes the mucus layer of the stomach which provides the ecological niche in the antrum, which is conducive for its habitations. The break down of mucus layer and damage to surface epithelial cells are the basis of ‘Leaking roof’ hypothesis of the pathogenesis of Helicobacter pylori.22

Regulation of gastric secretion:

Gastric motility and secretion are regulated by neural and hormonal mechanisms

a) The neural component: It comprises of;

1. Local autonomic reflexes involving cholinergic neurons.

2. Impulses from the CNS by the way of Vagus nerves.

b) The hormonal component: It involves various gastro intestinal hormones like gastrin, cholecystokinin and secretin.

Secretion of gastric juice has three interconnected phases:

1. Cephalic phase

2. Gastric phase

3. Intestinal phase

Cephalic phase:

Cephalic phase of gastric acid secretion acts by stimulating the vagal centre via the hypothalamus. Parietal and Chief cells are affected by direct cholinergic stimulation.

Gastric phase:

It starts by food entering and distending the stomach. Local and vasovagal distention reflexes stimulate the acid secretion of the stomach. Gastrin is released from the specialised ‘G’ cells of the antrum of stomach in response to food in the stomach and gastric distention. Gastrin then stimulates the acid secretion by the parietal cells in the body of the stomach.

Intestinal phase:

Gastric secretion is stimulated by food and its digestive products in the intestine. This may be due to stimulation of neuro-receptors and release of intestinal gastrin. In contrast acidification of the duodenum and the antrum results in inhibition of further acid secretion. This may be due to vagal inhibition or release of secretin or CCK-Pz(cholecystokininpancreozymin).

HISTORY & DEVELOPMENT OF ENDOSCOPY

As early as the 19th Century, attempts were been made to examine the interior of the upper GIT by reflecting light in to the body cavities through a hollow cylinder, but it was not until Thomas Edison's invention of the incandescent light bulb that it became possible in the late 1870's to perform rigid endoscopy.

Nevertheless progressively smaller lamps were developed that allowed insertion into the stomach through rigid endoscopes, but the nature of the light made it impossible to perform long or complex studies due to overheating of the instruments.

In addition the inability to adapt rigid instruments to the curvatures of the bowel permitted only limited examination of the upper GI tract.

These procedures were mostly performed by surgeons, such as the 19th Century Polish surgeon Johann Von Mikulicz-Radecki.

The era of flexible endoscopy began with the introduction of the semi rigid gastroscope by R.Schindler in 1936 through work developed in collaboration with the German physician Georg Wolf. The way to the development of a flexible fiberscope was paved by Baird's demonstration in 1928 that light and images could be transmitted through a single glass or quartz fiber.

In 1950's when Van Heel in the Netherlands and H.Hopkins and N.S.Kapany in England working independently, developed usable flexible glass fiber bundles that could transmit light across relatively long distance and into the body cavities.

The next phase of development took place in Ann Arbor at the University of Michigan, Physicians H.M.Pollard and Basil Hirschowitz , C.Wilbur Peters in collaboration with Physics students Lawrence Curtis, designed the first clinically useable, completely flexible endoscope.

Hirschowitz and Curtis started working on this concept in 1955 by developing an instrument composed of a bundle of individual glass fibres that was in theoretical capable of transmitting light as well as images.

Along the way, then encountered numerous problems such as fiber "Crosstalk", which differed the light, making interpretation of the images impossible.

This led to the invention of a glass coating for the fibres for insulation and to the development of fiber scope.

The first controllable tip gastroscope was developed in 1962 and in contrast to the most landmark inventions, was first applied clinically and then found an industrial application in the examination of jet engines.

After trying the flexible gastroscope on himself, Hirschowitz first used it in a patient with a bleeding duodenal ulcer in February 1957. The diagnosis was successfully established, and the patient underwent operation based on Hirschowitz observations.

The first commercially produced fiberoptic endoscope made by American cystoscope makers Inc, Norwalk, CT. was first used in 1961 and the results were published in the Lancet in may of that year.

Once the development and wide spread use of fiberoptic upper GI endoscope became a routine practice, the therapeutic potential was established. Experimental studies, such as those by W.D.Blackwood, S.Silivis, J.P.Papp, C.Sugawa and others demonstrated the feasibility and safety of endoscopic haemostasis.

This has paved the way for the use of endoscopes as vehicle for numerous accessories so that today endoscopic surgery include methods of haemostasis, excision, ablation, dilatation, decompression, sclerosis and foreign body removal.25

INSTRUMENTATION

Flexible endoscope come in a variety of diameter and lengths, either direct-viewing or video. The primary endoscope used for upper GI endoscopy is a zero degree, forward viewing endoscope, where as duodenoscope visualizes the GI tract at 90º to the shaft. Side viewing endoscope is primarily used in the duodenum to visualise the ampulla of Vater, but they may also be used in the stomach.

All endoscopes are either video or fibreoptic and all have a control head. In the fiberoptic units, an eye piece is present for either direct visualization or for video attachment.

The shaft of the endoscope is flexible, especially at the distal tip, which has deflection capabilities ranging from 90-240 in the up/down position and 100 in right or left directions. The diameter of the insertion tube can range between 5.5mm to the distal tip to 11mm for a therapeutic endoscope. The diameter of insertion tube for duodenoscope ranges between 11.5 to 12.5mm.

The controls for maneuvring the deflection tip are located on the control head with a large inner knob producing up or down deflection and the smaller outer knob producing a left or right deflection. Two depressable buttons are located adjacent to these deflection knobs and when pressed the top button produces suction that may be necessary during the examination. The lower button serves two additional functions. Air insufflations occurs by simple placement of a finger over the button without applying pressure. When this button is depressed a small amount of water is released.From the tip of the endoscope that is useful for cleaning the tip during the examination if it becomes dirty.

In the video endoscope, video control buttons on the top of the control head are used to freeze an image on the video screen or to save the image for printing.The flexible shaft is usually 110-120cms in length. This endoscope contain a working channel that varies between 2mm (paediatric endoscope) to 3.7mm(therapeutic endoscope). On the other hand the instrument channel in the duodenoscope varies from 3.2-4.2mm.

Biopsy forceps, cytology brushes, or other diagnostic instruments are passed through the accessory channel. A double lumen therapeutic endoscope is also available for more advanced therapeutic endoscopy.

The flexible endoscope is connected to a light source that is either 300W Xenon arc lamp or a halogen- tungsten lamp. In addition, air and water pumps for insufflations, suction and irrigation are connected to the endoscope via the light source unit and controlled using the control buttons.

If a video monitor is being used, this is also connected to the endoscope through the light source.

Proper hand positioning and manipulation of the flexible endoscope is key to perform an efficient examination. Most endoscopists will hold the control head of the endoscope in the left hand, with the thumb on the up/down knob and the index and middle finger on the suction and air/water button. The thumb & index finger are then used to control the deflection tip during examination. The right hand of the endoscopist is used to hold the flexible shaft for insertion, withdraw and rotation during the examination.26

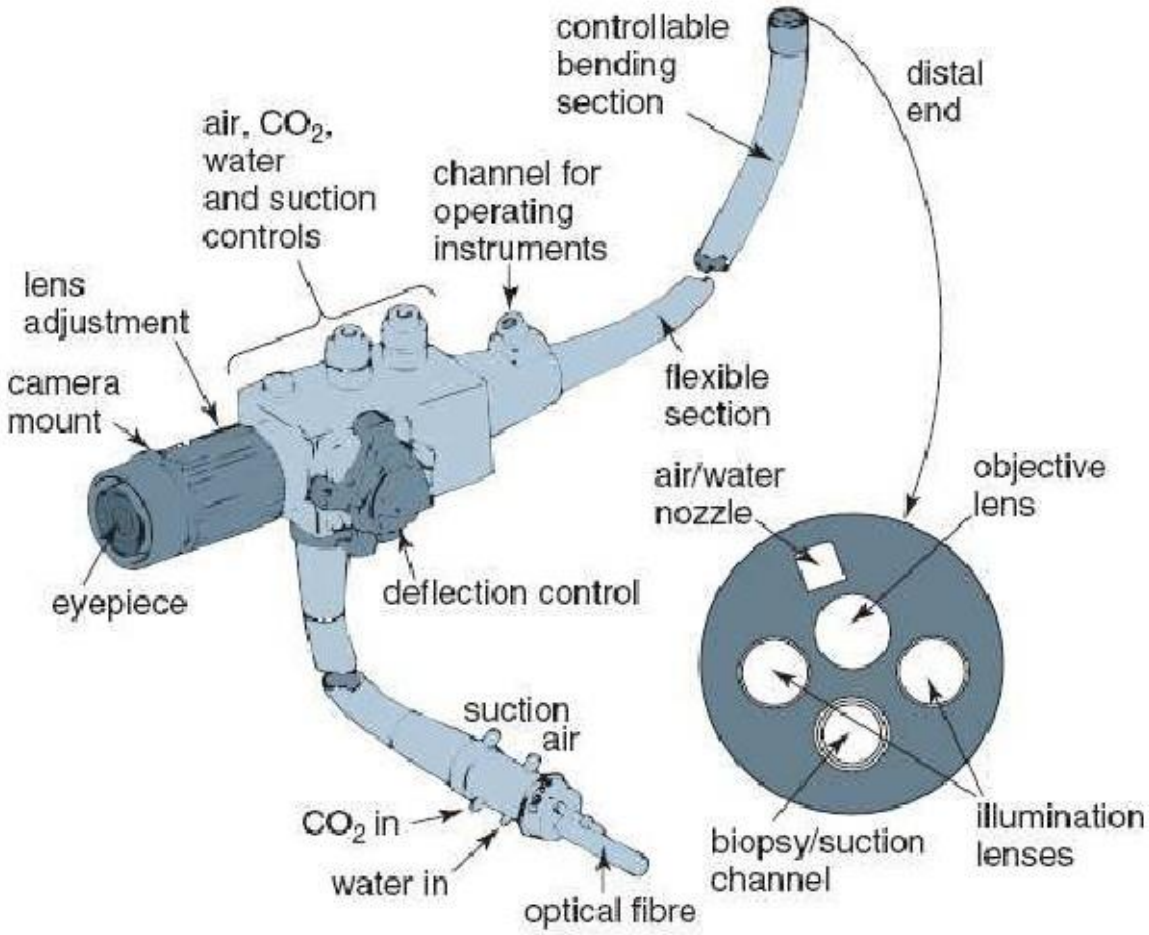


Fig 6. Parts of the endoscope

UPPER GASTROINTESTINAL ENDOSCOPY

PROCEDURE:

PRINCIPLES:

Upper GI endoscopy plays a dominant role in the examination of the upper GI tract. It provides both direct & complete visualization of the area and direct access for tissue sampling and/or therapeutic intervention.

This should be mastered by any clinician with a special interest in diseases of the esophagus, stomach and duodenum.

PATIENT PREPARATION:

The procedure is explained to patient in simple terms. During the clinical evaluation , allergies, current medication and previous medical history are reviewed, the need for antibiotic prophylaxis is assessed.

The patient should fast over night before the procedure. Out patients should be accompanied, particularly if intra venous sedation is to be used.

Having a calm & relaxed patient avoids to some extent the need for sedation. A tense patient should not be submitted to endoscopy under simple topical anaesthesia. Proper sedation dictates the use of pulse oximetry and ECG. A Lignocain gargle or spray is used for topical anaesthesia of the pharynx and hypopharynx.

When needed,adequate sedation may be obtained with benzodiazepines (diazepam, midazolam). Pethedine hydrochloride may be added for relaxation and analgesia. This medication should be administered slowly in small doses until the desired level of sedation is obtained.

TECHNIQUE

INTRODUCTION OF THE ENDOSCOPE:

The patient lies in the left lateral decubitus position. Following appropriate topical anaesthesia, a mouth piece is postioned between upper and lower teeth. Endoscope is advanced, taking care to stay on the midline and at the interface between the tongue and hypopharyngeal mucosa. Tongue, uvula, epiglottis and cricoarytenoid cartilages are seen. Passing beside the midline, the cricoarytenoid cartilages are passed and the tip of the endoscope stops on the cricopharyngeus. Gentle local pressure while asking the patient to swallow allows the tip of the endoscope to pass into the cervical esophagus.

EXAMINATION OF ESOPHAGUS:

The instrument is advanced under direct vision, with the tip of the endoscope always central in the lumen. Using the optimal insufflations to keep the lumen of the esophagus well distended.

First hand inspection is important, because no trauma has caused by the manipulation or passage of the instrument.

Two rules should always be followed:

1. Endoscope must advanced with clear vision of the central lumen.

2. If direct vision is obscured or there are any doubts, the endoscope should be withdrawn.

Land marks distal to cricopharyngeal sphincter are extra luminal compression of left main bronchus, aortic arch and pulsations of left heart in the distal half.

The gastro-esophageal mucosal junction is usually identified at 38-40cms from the incisors. This junction is usually serrated and readily identified by the color difference between the esophageal and gastric mucosa, called as Z line.

The position of the esophageal hiatus in the diaphragm is identified by asking the patient to inhale deeply, the diaphragmatic hiatus during inspiration creates an imprint on the esophageal and gastric wall. The position of both the hiatus and the mucosal junction are recorded in order to document the possibility of a hernia or of a columnar lined esophagus.

PASSAGE IN TO THE STOMACH:

Gastro-esophageal junction should be observed for closed or widely patulous. Passage in to the gastric lumen is usually a simple manoeuvre that occurs without resistance.

On entering the stomach, it becomes distended with air and this often causes discomfort to the patient. By tipping the end of the endoscope slightly down and towards the left, a view of greater curvature and of the posterior wall is obtained. Aspiration of all retained liquid is done to reduce the risk of aspiration & to allow proper examination of the stomach. A rotation movement of the tip of the instrument allows examination of the anterior and posterior walls of the body of the stomach. The lesser curvature down to the angulus and the greater curvature are viewed by the same position motion. The most proximal part of both curvatures are better examined when using the J manoeuvre.

By rotating and angulating the tip endoscope is advanced to assess the antrum. Prepyloric and pyloric ring observed directly, the passage through the pylorus being done under direct vision. When the pylorus yields, complete assessment of the first part of the duodenum is done as far as the superior duodenal angle. While the tip of the endoscope lies along the distal lesser curvature and while the stomach is distended, rotation of the instrument is

accomplished towards the greater curvature, complete 180 degree upwards angulation of the endoscope tip completes the J manoeuvre.

The endoscope is pulled back while the stomach is distended, from the cardia to angulus. swinging of the retroflexed tip allows proper visualization of the stomach. Simultaneous rotation of the endoscope gives excellent view of the lesser curvature

After straightening the tip endoscope is gently pulled back examining the esophagus again. Patients are encouraged to avoid drinking or eating for approximately 30mins after the procedure.26

ENDOSCOPIC BIOPSY:

Typical lesions routinely evaluated by biopsy are esophageal strictures, mass lesions, gastroduodenal ulcers, gastroduodenitis and polyps. Diagnostic yield increases when multiple specimens are taken of any suspicious lesion, if one suspects a malignancy, six biopsy specimens and cytology will increase diagnostic accuracy to better than 90-95% lesions arousing suspicion for being varices should not be biopsied, as this can lead to significant bleeding.

Biopsy forceps come in many shapes and sizes. The biopsy instrument is passed through the working channel, and once the tip is visible the jaws are opened, pushed into the mucosa, closed and then quickly pulled back, bringing

with them an adequate specimen. Biopsy forceps containing a spike can be used to obtain multiple specimens without having to remove it from the endoscope.

Biopsies for gastric ulcers should typically be taken in all four quadrants and at the base of the ulcer. The transition zone between the ulcer and surrounding mucosa is the area that most likely contains increased mitotic activity in malignant ulcers and therefore biopsy of this region improves diagnostic yield.

Biopsies of submucosal masses can have limited yield because the submucosal location is not easily reached. To increase yield several biopsies should be taken. Caution is the rule – because the area can become weakened and be at risk for perforation.

Esophageal stricture, such as demonstrate dysplasia or malignant transformation and specimen obtained. Polyps in the stomach or duodenum can be cancerous and should be sampled, either hot or cold biopsy forceps can remove diminutive polyps less than 5mm in diameter. Whereas a snare is best for larger polyps. The snare is placed at the base of a pedunculated polyp, and the polyp is removed in piecemeal fashion.

Japanese investigators have developed technique for lesion removal where by a suction apparatus is passed through the endoscope and lesion is grasped with suction. A snare is then placed around the base of the lesion &

closed tightly and removal of the specimen is possible. If significant bleeding results, standard coagulation technique can be employed.26

PROGRESS OF INTRAGASTRIC OBSERVATION THROUGH THE FIBEROSCOPE:

1. Simplification of the technique of intra gastric observation based on direct vision.

2. Elimination of blind spots.

3. Regulation of various endoscopic conditions.

4. Advance in observing fine changes through close up observation.

5. Improvement of recording ability by aiming recording photography equipment.

6. Revolutionizing the technique of biopsy on direct vision through the use of fiberscope.

7. Progress in diagnosing cancer cells by viewing cells according to the direct vision method with the fiberscope.

8. Precise observation through the application of supplemental techniques,such as washing the lesion and applying a pigment solution..

DYSPEPSIA: Definition and prevalence

Dyspepsia (from the Greek (Dys-), meaning hard or difficult, and (Pepse), meaning digestion) is chronic or recurrent pain or discomfort centered in the upper abdomen.28,29 Discomfort, in this context, includes mild pain, upper abdominal fullness and feeling full earlier than expected with eating. It can be accompanied by bloating, belching, nausea or heartburn. It may be called indigestion. Heartburn is excluded from the definition of dyspepsia in ICD-10, as it usually has a different cause and management pathway. When a patient has dyspepsia, but no underlying disease is found, the patient is said to have non-ulcer dyspepsia or functional dyspepsia or idiopathic dyspepsia.

Classification:

Dyspepsia has been proposed to have symptomatic subgroups.30,31,32

- Ulcer like - Pain centered in the upper abdomen is the predominant (most bothersome) symptom.
- Dysmotility like - An unpleasant or troublesome non-painful sensation (discomfort) centered in the upper abdomen is the predominant symptom; this sensation may be characterized by or associated with upper abdominal fullness, early satiety, bloating, or nausea.
- Reflux like
- Nonspecific.

Dyspepsia occurs in 40% of the population, leads to General practitioner consultation in 5% and referral for endoscopy in 1% of the population annually. In patients with signs or symptoms severe enough to merit endoscopy, 40% have functional or non-ulcer dyspepsia, 40% have gastro-esophageal reflux disease and 13% have some form of ulcer.33 Alarm features or red flags that may indicate serious underlying diseases are:34

- Age older than 55 years with new-onset dyspepsia
- Family history of upper gastrointestinal cancer
- Unintended weight loss
- Gastrointestinal bleeding
- Progressive dysphagia
- Odynophagia
- Unexplained iron-deficiency anemia
- Persistent vomiting
- Palpable mass or lymphadenopathy
- Jaundice

ETIOLOGY:

Etiology can be broadly classified into 2 main groups34

Structural abnormalities.

Functional (Non ulcer) dyspepsia.

STRUCTURAL ABNORMALTIES:

Hiatus hernia.

Gastro-esophageal reflux disease (GERD).

Barrett's esophagus.

Peptic ulcer disease.

Esophageal, gastric and duodenal cancer.

HIATUS HERNIA:

A hiatus hernia occurs when part of the stomach moves up in to the chest through a defect in the diaphragm. It is a common problem occurring in 10% of people and the hernia rarely causes symptoms on its own. The presence of a hiatus hernia can cause weakness of the lower esophageal sphincter and this in turn can cause reflux of the acidic stomach contents into the esophagus.

This causes the sensation of heartburn and patients with a hiatus hernia are more prone to heartburn than those without this defect. Nevertheless it is important to emphasise that not all patients with hiatus hernia have heartburn and some patients with heartburn do not have a hiatus hernia.

GASTRO-ESOPHAGEAL REFLUX DISEASE (GERD):

Gastro-esophageal reflux may occur when the pressure of the high-pressure zone in the distal esophagus is too low to prevent gastric contents from entering the esophagus or when a sphincter with normal pressure undergoes spontaneous relaxation, not associated with a peristaltic wave in the body of the esophagus. GERD is often associated with a hiatus hernia.

The most common presentation of patients with GERD is a long-standing heartburn and a shorter history of regurgitation. Heartburn, when typical, is a very reliable symptom. Heartburn is confined to the epigastric and retrosternal areas. It is identified as a caustic or stinging sensation. It does not radiate to the back and is not characteristically described as a pressure sensation.

BARRETT'S ESOPHAGUS:

In some patients, prolonged acid and perhaps alkaline injury leads to a change in the esophageal mucosa from its usual squamous epithelium to a columnar configuration (Barrett's esophagus). The cells almost always extend proximally from the squamo columnar junction in a contiguous pattern.

If Barrett's esophagus is found,multiple biopsies are necessary to exclude dysplasia, which may indicate a tendency toward the development of adenocarcinoma.

Although the incidence of adenocarcinoma in patients with Barrett's esophagus is about 40 times greater than that in the general population (Barrett's studies with increased incidence), the incidence of cancer in these patients is still very low.

Because Barrett's esophagus arises from gastro-esophageal reflux injury (of acid or bile), Long segment Barrett's esophagus, diagnosed when at least 3cm of the distal esophagus is lined by columnar epithelium, has greatest malignant potential and surveillance is recommended for this disorder. Short segment Barrett's esophagus, for less than 3cm of columnar lined esophageal mucosa, is thought to have a lower malignant potential and the role of surveillance is uncertain. Although no columnar lining may be visible, intestinal metaplasia may be found in biopsies taken at the gastro-esophageal junction. While 20% of the population have evidence of intestinal metaplasia at the gastro-esophageal junction, again the malignant potential of this lesion is uncertain and surveillance is not recommended.

PEPTIC ULCER DISEASE (PUD):

Ulcer is caused by acid peptic digestion of the mucosa to variable depth either in mucosa containing acid secreting cells or in other sites. Peptic ulcer extends through the muscularis mucosa, an erosion is superficial to the muscularis mucosa.

Although the name suggests an association with pepsin, it is the acid which is important for the occurrence of peptic ulcer. May be acute ulcers which are shallow and multiple or chronic which are single, deep and scirrhous.

Common sites:

1. 1st part of duodenum
2. Lesser curve of stomach
3. Prepyloric and pyloric channel

Gastric ulcer:

Seen commonly in late middle age and the incidence increases with age.

Sex incidence is found to be equal.

Duodenal ulcer:

Most common in middle age, more common in males. Male to female ratio was found to be 3:1. 10 - 20% of patients with a gastric ulcer may have concomitant duodenal ulcer.

Etiology:

1. Helicobacter pylori infection

2. Endocrine –

a) Zollinger-Ellison syndrome b) Cushings syndrome

c)Parathyroid tumour - hypercalcemia

3. Genetic: cases with blood group ‘O’

4. Drugs : NSAIDS, aspirin, steroids

5. Smoking:

a) Predispose to ulcer formation

b)Increases the relapse rate after treatment.

6. Alcohol7. Diet: irregular diet, spicy food and excessive intake of coffee and tea provoke the formation of peptic ulcer.

8. Emotional factors: anxiety, stress have always been incriminated to cause peptic ulcer.

Pathogenesis:

1. Loss of mucosal defense with hyperacidity

2. Gastric mucus is an important barrier that protects the gastric mucosa from the effects of acid and pepsin.

3. Decreased bicarbonate concentration

4. Decreased gastric mucosal prostaglandin production

5. Acid overproduction is an important factor for causing DU.

H.pylori:

It is the most important factor in the development of peptic ulcer. Fifty percent of the world's population is infected with H. pylori, a major cause of chronic gastritis. Helicobacter also clearly has an etiologic role in the development of gastric lymphoma. H.pylori is a small curved, motile, Gram negative, microaerophilic rod with multiple polar flagellae. In stomach it remains close to the gastric mucus secreting cells.

It hydrolyses urea → ammonia → increased gastrin.

ESOPHAGEAL AND GASTRIC CANCER:

Gastric and esophageal cancers are rare, accounting annually for 1% of deaths from all causes. Gastric cancer is on the decline, while esophageal cancer is on the increase. Gastric cancer may be declining because of the decreasing prevalence of H.pylori. Squamous cell carcinoma and adenocarcinoma account for 95% of all esophageal tumours. Traditionally squamous carcinoma was the most frequent lesion but in recent years adenocarcinoma has become the predominant disease.

Adenocarcinoma of the esophagus is believed to originate from columnar metaplasia of the esophagus (Barrett's esophagus), providing a rationale for endoscopic screening of patient's with Barrett's esophagus.Adenocarcinoma is responsible for over 95% of all gastric malignancies. Half of patients are inoperable at the time of diagnose and few of these survive five years,while of those undergoing operative treatment 20% are alive after 5 years. Overall 5 year mortality for this disease is therefore approximately 90%. Gastric neoplasia is strongly associated with H.pylori infection but as the vast majority of H.pylori infected individuals do not develop gastric carcinoma other environmental and genetic factors must be important.33

FUNCTIONAL (NON ULCER) DYSPEPSIA:

Functional gastrointestinal disorders include a variable combination of chronic or recurrent gastrointestinal symptoms that do not appear to be explained by structural or biochemical abnormalities. These functional disorders include symptoms attributed to dysfunction of the oropharynx, esophagus, stomach, small bowel, large bowel and biliary tract.

Non-ulcer dyspepsia is a heterogeneous syndrome. It has been proposed that this entity can be subdivided into a number of symptomatic clusters or groupings that suggest possible underlying pathogenetic mechanisms. These groupings include ulcer-like dyspepsia (typical symptoms of peptic ulcer are present), dysmotility (stasis)- dyspepsia (symptoms include nausea, early satiety, bloating, and belching that suggest gastric stasis or small intestinal dysmotility), and reflux-like dyspepsia (heartburn or acid regurgitation accompanies upper abdominal pain or discomfort).likely a multifactorial disorder. Motility abnormalities may be important in a subset of dyspepsia patients but probably do not explain the symptoms in the majority.

Epidemiological studies have not convincingly demonstrated an association between Helicobacter pylori and The etiology of non-ulcer dyspepsia is not established, although it is non-ulcer dyspepsia. Other potential etiological mechanisms, such as increased gastric acid secretion, psychological

factors, life-event stress, and dietary factors, have not been established as causes of non-ulcer dyspepsia.34

OTHER CAUSES:

1. Biliary or pancreatic diseases.

2. Metabolic disturbances.

3. Irritable bowel disease.

4. Psychiatric diseases.

INVESTIGATIONS:20

UPPER GI ENDOSCOPY: Endoscope is used to visualize the esophagus, stomach and proximal duodenum, if necessary therapeutic procedures can be perform.

Endoscopy has now become the gold standard test for detecting esophageal, gastric and duodenal lesions.

Investigations for H.pylori infection:

Tests for Helicobacter pylori may be divided into those that require endoscopy (invasive tests) and those that do not require endoscopy (non-invasive tests).

Invasive tests:

a. Histology

b. Culture

c. Rapid urease test (RUT)

d. Cytology

e. Polymerase chain reaction (PCR)

Non-invasive tests:

a) Serology.

b) Urea breath tests.

c) Faecal antigen testing.

TREATMENT

1. Reassurance.

2. Pharmacological treatment:

a) H 2 receptor blockers- Ranitidine 150mg bid.

b) Proton pump inhibitors- Omeprazole 20mg, Rabeprazole 20mg,

Pantoprazole 40mg.

c) Antacids and alginates- Aluminium hydroxide, Magnesium trisilicate,

Dimeticone and Peppermint oil.

d) Prostaglandin analogues- Misoprostol.

e) Prokinetics- Domperidone and Cisapride

SURGICAL PROCEDURES: 21

The discovery of H.pylori and the development of powerful acid suppressive therapy have revolutionized the medical therapy of peptic ulcer and gastro- esophageal reflux disease. This has made peptic ulcer surgery almost obsolete.

Anti-reflux surgery is reserved for selected patients with documented acid reflux whose symptoms are unresponsive to medical therapy or who do not wish to take long termPPI treatment.

ANTI-REFLUX SURGERY

FUNDOPLICATION (OPEN OR LAPROSCOPIC APPROACH)

a) Nissen fundoplication (360- degree wrap)- most common anti-reflux surgery.

b) Partial anterior fundoplication.

c) Partial posterior fundoplication.

ENDOSCOPIC THERAPY:

Recently, several endoscopic techniques have been developed for the treatment of GERD. These procedures have sparked significant interest because they each promise a mechanical treatment for reflux with less invasion than a fundoplication.

These techniques attempt to augment the LES by suturing, radiofrequency energy, Plexiglas injection or biocompatible polymer injection.

PEPTIC ULCER SURGERY

a) TRUNCAL VAGOTOMY: Division of both vagus nerves above the hepatic & celiac branches just above the GE junction. This procedure is usually combined with drainage procedure.

- Gastrojejunostomy
- Pyloroplasty

b) SELECTIVE VAGOTOMY: Division of both vagus below the hepatic & celiac branches.

c) HIGHLY SELECTIVE VAGOTOMY (HSV):Also called parietal cell or

proximal gastric vagotomy. Severs vagal nerve supply to proximal 2/3rd of the stomach and preserves vagal innervation to the antrum and Recurrence rate 5 to 10%. pylorus.

GASTROJEUJUNOSTOMY: Ananstomosis between proximal jejunum and the most dependant portion of greater curvature of the stomach. Anastomosis is antecolic / retrocolic, isoperistaltic, no loop, no tension.

VAGOTOMY + ANTRECTOMY (This procedure has got the lowest recurrence rate < 2%).

Billroth I reconstruction (Gastroduodenostomy).

Roux-en-Y Gastrojejunostomy.

PYLOROPLASTY:

a) Heineke-Mikulicz pyloroplasty involves a longitudinal incision of the pyloric sphincter followed by a transverse closure. Most commonly performed pyloroplasty.

b) The Finney pyloroplasty is performed as a gastroduodenostomy with division of the pylorus.

c) The Jaboulay pyloroplasty differs from the Finney procedure in that the pylorus is not transected.

SURGERY FOR GASTRIC CANCER:

1.Endoscopic mucosal resection (EMR).

2.Endoscopic submucosal dissection (ESD).

3.Wedge resection.

4.Open gastrectomy (Partial/ subtotal).

5.Laparoscopically assisted gastrectomy (Partial/ subtotal).

LYMPH NODE LEVELS:

N 1 : Peri gastric nodes.

N 2 : Nodes along the vessels.

N 3 : Distant nodes.

EXTENT OF LYMPHADENECTOMY:

- D 1 Resection: Removal of tumour and N1 nodes.

- D 2 Resection: Removal of tumour and N1, N2 nodes also removes the peritoneal layer over the pancreas and anterior mesocolon.

SURGERY FOR ESOPHAGEAL CANCER: ESOPHAGECTOMY

The Trans-hiatal Approach: The trans-hiatal esophagectomy is performed through an upper midline laparotomy and left cervical incision.

The Ivor Lewis Approach: The trans-abdominal, trans-thoracic approach.

Three-Field Esophagectomy: This approach is carried out through separate laparotomy, right thoracotomy, and cervical incisions.

The Thoracoabdominal Approach: The left thoracoabdominal approach is probably the least utilized of all approaches to the esophagus.

The Minimally Invasive Approach: A number of minimally invasive techniques laparoscopic, esophagectomy. To esophagectomy, hand-assisted,have been described. And These include thoracoscopic, robotic-assisted

METHODOLOGY

A prospective clinical study was undertaken at general surgery department, RajivGandhi Government General Hospital attached to Madras Medical College , to know the various upper gastro-intestinal endoscopic findings in patients presenting with late onset dyspepsia at RGGGH, Chennai". The study was conducted over a period of 6 months(June 2013 to Nov 2013). The patient selection was by convenience sampling.

Dyspeptic patients were included in this study with their informed consent. A detailed clinically history was elucidated, followed by careful clinical examination, which were recorded as per the proforma. All the patients included in the study underwent upper gastrointestinal endoscopy and the findings were noted.

INCLUSION CRITERIA:

1) patient age above 40yrs with dyspeptic symptoms.

2) Patient with alarm symptoms.

3) Patient with previously diagnosed and treated cases of gastric ulcer, duodenal ulcer, complicated peptic ulcer, coming with dyspeptic symptoms.

EXCLUSION CRITERIA:

1) Patients who are endoscopy negative but have dyspepsia, may have motility disorders.

2) Individuals who had an endoscopy indications other than dyspepsia.

3) Not willing for endoscopy.

PROCEDURE:

All the patients in this study group, on outpatient basis underwent upper gastro-intestinal endoscopy under topical anesthesia. The patients were asked to fast for 12 hours prior to the procedure. Only a few patients were given 5-10mg diazepam intravenously for sedation.

Lignocaine viscous or oral lignocaine sprays were given to the patient 5-10 minutes before the procedure for the local anaesthetic effect. The upper gastro-intestinal endoscopy was conducted with flexible, fibreoptic endoscope with patients in left lateral positions.

The instrument is advanced under direct vision, with the tip of the endoscope in central lumen. Using the optimal insufflations to keep the lumen of the esophagus well distended. Esophagus was looked for any inflammatory changes, growth. The gastro-esophageal mucosal junction was identified at 38-40cms from the incisors.(This junction is usually serrated and readily identified by the color difference between the esophageal and gastric mucosa, called as Z line).

The position of the esophageal hiatus in the diaphragm is identified by asking the patient to inhale deeply, the diaphragmatic hiatus during inspiration creates an imprint on the esophageal and gastric wall. The position of both the

hiatus and the mucosal junction are recorded in order to document the possibility of a hernia or of a columnar lined esophagus.

Gastro-esophageal junction should be observed for closed or widely patulous. On entering the stomach, endoscope slightly down and towards the left, a view of greater curvature and of the posterior wall is obtained.

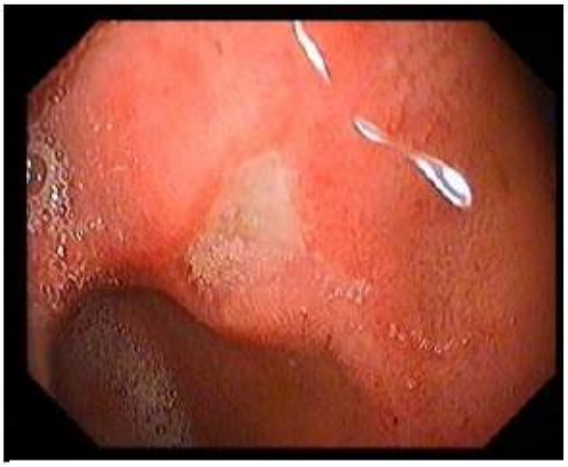
Aspiration of all retained liquid is done to reduce the risk of aspiration and to allow proper examination of the stomach. A rotation movement of the tip of the instrument allows examination of the anterior and posterior walls of the body of the stomach. The lesser curvature down to the angulus and the greater curvature are viewed by the same position motion. The most proximal part of both the curvatures are better examined when using the J manoeuvre. Stomach was looked for inflammatory changes, ulcer, growth.

By rotating and angulating the tip endoscope is advanced to assess the antrum. Prepyloric and pyloric ring observed directly, the passage through the pylorus being done under direct vision. When the pylorus yields, complete assessment of the duodenum is done upto second part.

Two endoscopic biopsies were taken, 2 each from the gastric antrum and the abnormal looking area. The edge of the ulcer crater depending on the findings. The biopsies from the body and the antrum were randomly taken in cases where in the endoscopic findings were normal.

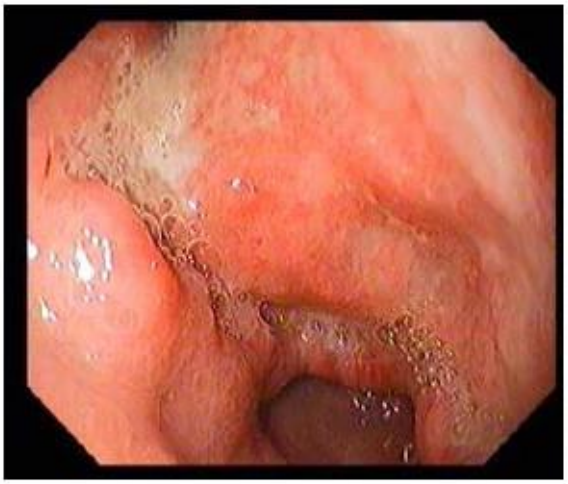
Flexible Optic Endoscopy:

Endoscopic view of gastric ulcer



Esophageal carcinoma

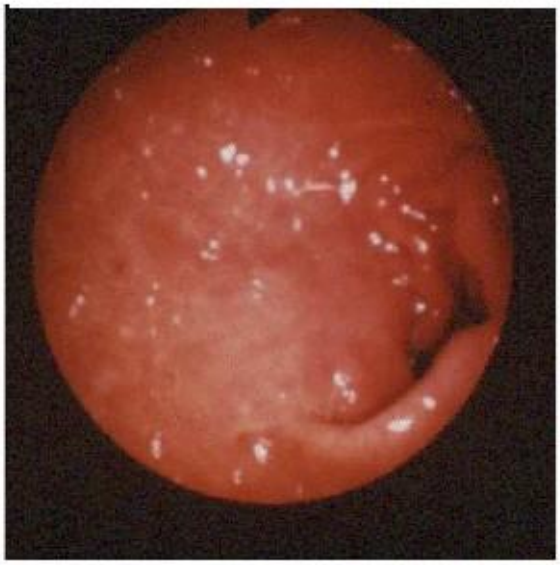
Gastric ulcer with carcinoma



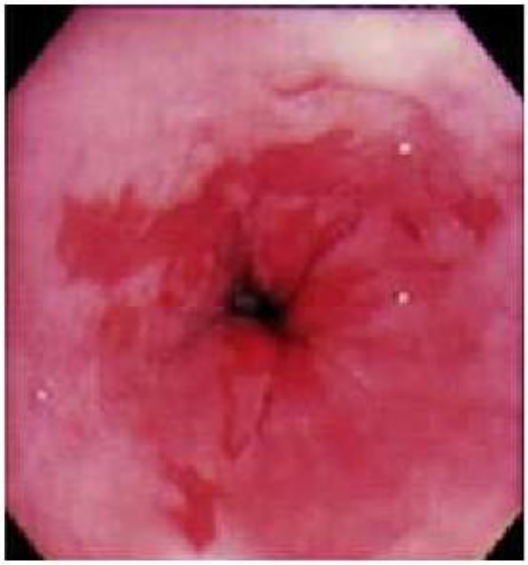
Antral gastric ulcer

Gastric antral gastritis

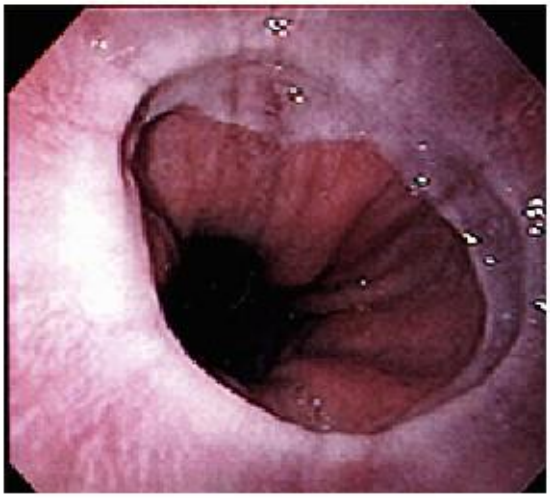
bile reflex gastritis



Endoscopic view of duodenal ulcer



Endoscopic view of Barrett's



Endoscopic view of Lax LES

(hiatus hernia)

Endoscopic biopsy instrument

Endoscopic biopsy

RESULTS

Out of 200 patients, there were 118 (59%) male patients, 82 (41%) female patients, age ranging from 40 years to 80 years. The mean age of the patients in this study was found to be 60 years.

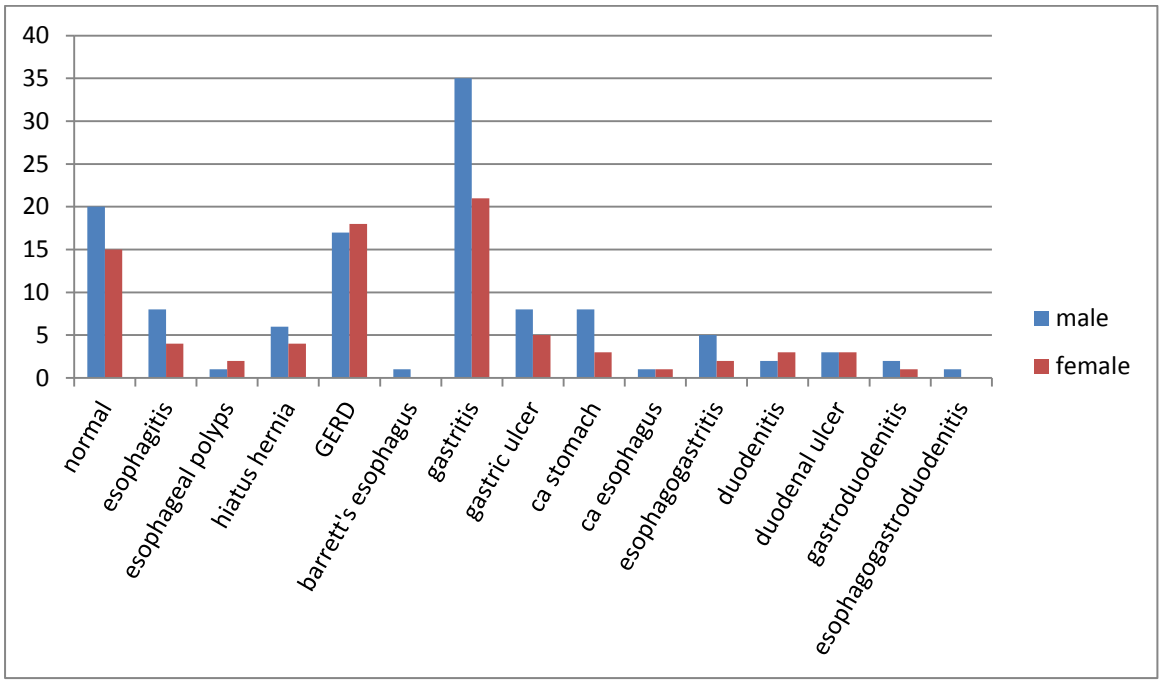
All these patients presented to our hospital with symptoms of dyspepsia for 4 or more than 4 weeks. Upper GI endoscopy was done in all patients.

Table-1 Various endoscopic finding in patients with dyspepsia

s.no	Endoscopic finding	Male(%)	Female(%)	Total(%)
1	Normal	20	15	35(17.5%)
2	Esophagitis	8	4	12(6%)
3	Esophageal polyp	1	2	3(1.5%)
4	Hiatus hernia	6	4	10(5%)
5	GERD	17	18	35(17.5%)
6	Barrett's esophagus	1	0	1(0.5%)
7	Carcinoma esophagus	1	1	2(1%)
8	Gastritis	35	21	56(28%)
9	Gastric ulcer	8	5	13(6.5%)
10	Carcinoma stomach	8	3	11(5.5%)
11	Esophagogastritis	5	2	7(3.5%)
12	Duodenitis	2	3	5(2.5%)

13	Duodenal ulcer	3	3	6(2%)
14	Gastroduodenitis	2	1	3(1.5%)
15	Esophagogastroduodenitis	1	0	1(0.5%)
	Total	118(59%)	82(41%)	200

Graph 1: Frequency of various diseases on endoscopy in patients presenting with dyspepsia



Normal study was observed in 35(17.5%) patients. Most common abnormal endoscopic finding was gastritis 56(28%) patients, followed by GERD in 35 (17.3%) of patients, esophagitis, hiatus hernia and duodenal ulcer,

which were present in 8(5.6%) patients each. Duodenitis in 5 (2.5%) patients, esophagogastritis and gastric ulcer were seen in 17 (8.5%) patients each.

Carcinoma stomach in 11 (6.5%) patients, gastroduodenitis in 3 (1.5%) patients, carcinoma esophagus in 2(1%) patients were noted Esophageal polyp, esophagogastroduodenitis and Barrett's esophagus present in 5 (2.5%) patient each, were the least common findings.

AGE DISTRIBUTION;

Table 2: Incidence of dyspepsia in different age groups

Age groups	No of cases(%)
41-50	39.5%
51-60	32 %
61-70	19%
71-80	9.5%

Graph 2. Incidence of dyspepsia in different age group

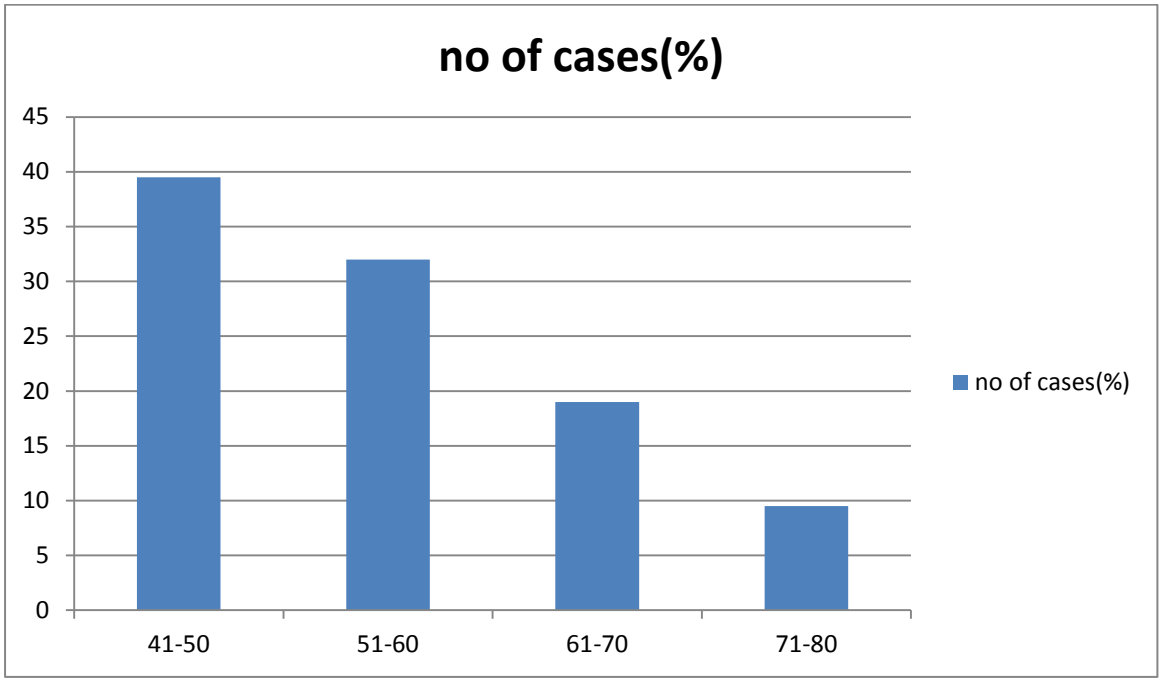
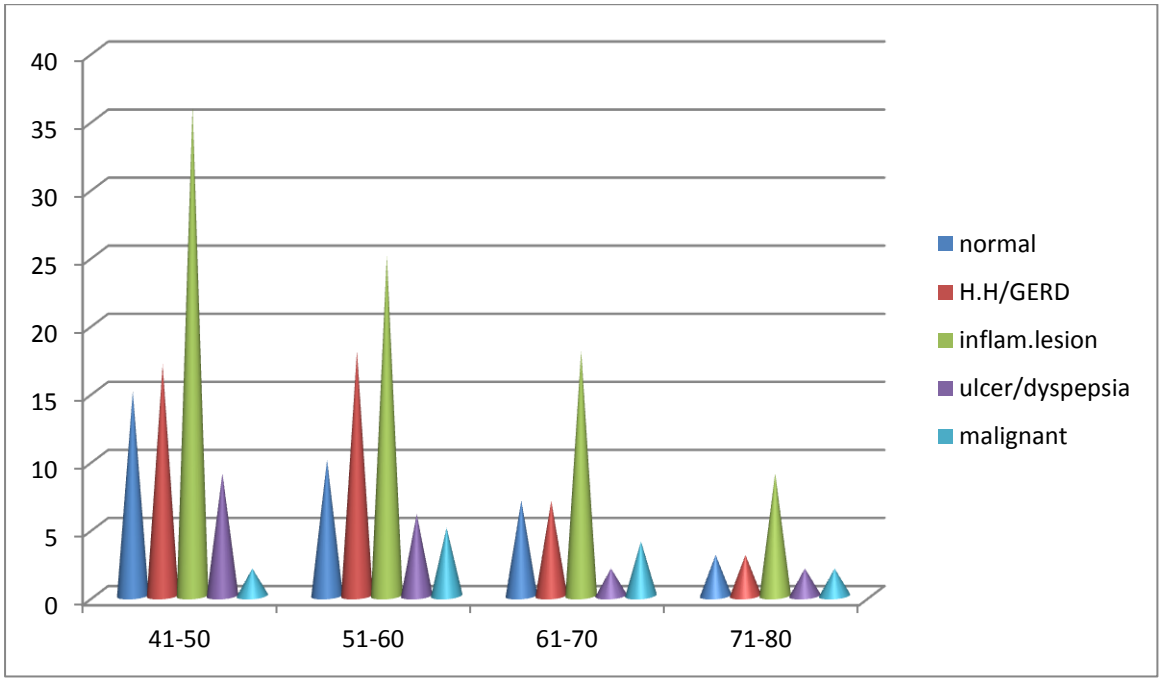


TABLE 3: Frequency of various diseases on endoscopy in different age group

Age Groups	Normal study	H.H/ GERD	Inflm lesion	malig	Ulcer/ dyspepsia	Total	percentage
41-50	15	17	36	2	9	79	39.5%
51-60	10	18	25	5	6	64	32%
61-70	7	7	18	4	2	38	19%
71-80	3	3	9	2	2	19	9.5%
TOTAL	35	45	88	13	19	200	100

Graph 3: Frequency of various diseases on endoscopy in different age group



All patients were subdivided into different age groups.

-Most common clinically significant endoscopic findings were seen in age group between 41-60 years.

-Hiatus hernia GERD were commonly seen in the age group between 41-60 years.

-Inflammatory lesions (gastritis, esophagitis, eso polyp, Barrett's esophagus, esophagogastritis, duodenitis, gastroduodenitis and

esophagogastroduodenitis) were commonly seen in the age group between 41-50 years.

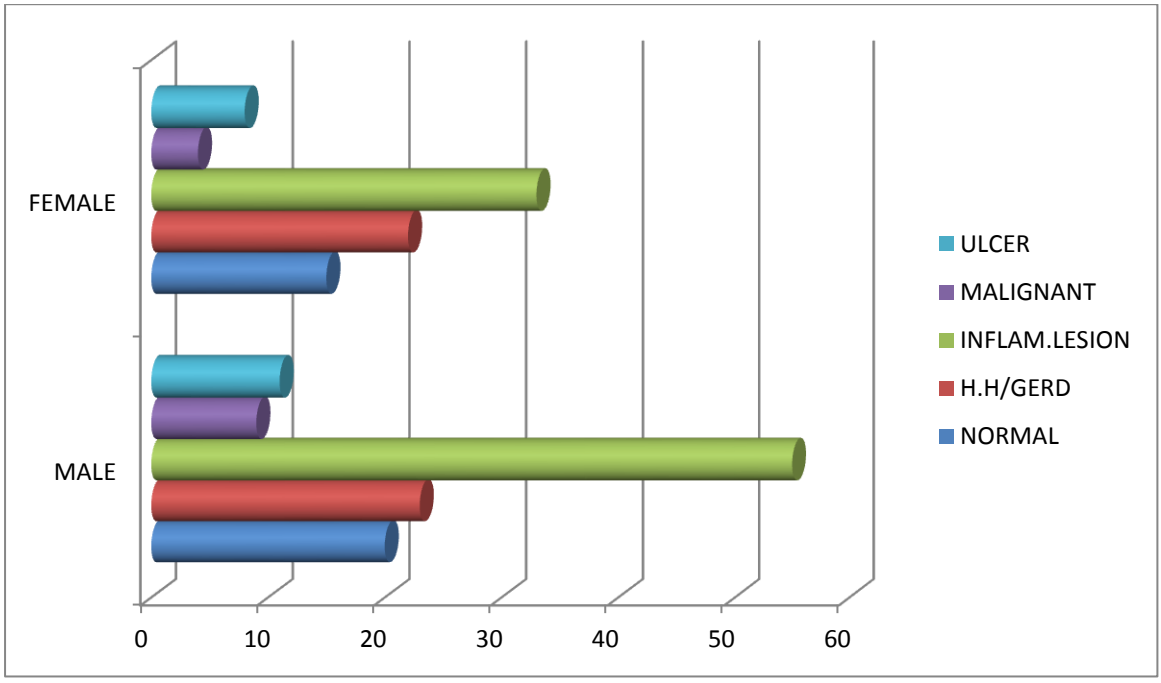
-Ulcer dyspepsia were commonly seen in the age group between 41-60 years.

-Malignant lesions were seen frequently in patients aged more than 50 years.

Table 4: Frequency of various diseases on endoscopy in males and females

GENDER	NORMAL STUDY	H.H/ GERD	INFLA LESION	MALIG	ULCER	TOTOL	PERCEN TAGE
MALE	20 (16.9%)	23 (19.5%)	55 (46.6%)	9 (7.6%)	11 (9.3%)	118	59%
FEMALE	15 (18.3%)	22 (26.8%)	33 (40.2%)	4 (4.8%)	8 (9.7%)	82	41%
TOTAL	35 (17.5%)	45 (22.5%)	88 (44%)	13 (6.5%)	19 (9.5%)	200	100%

Graph-4 Frequency of various diseases on endoscopy in different males and females

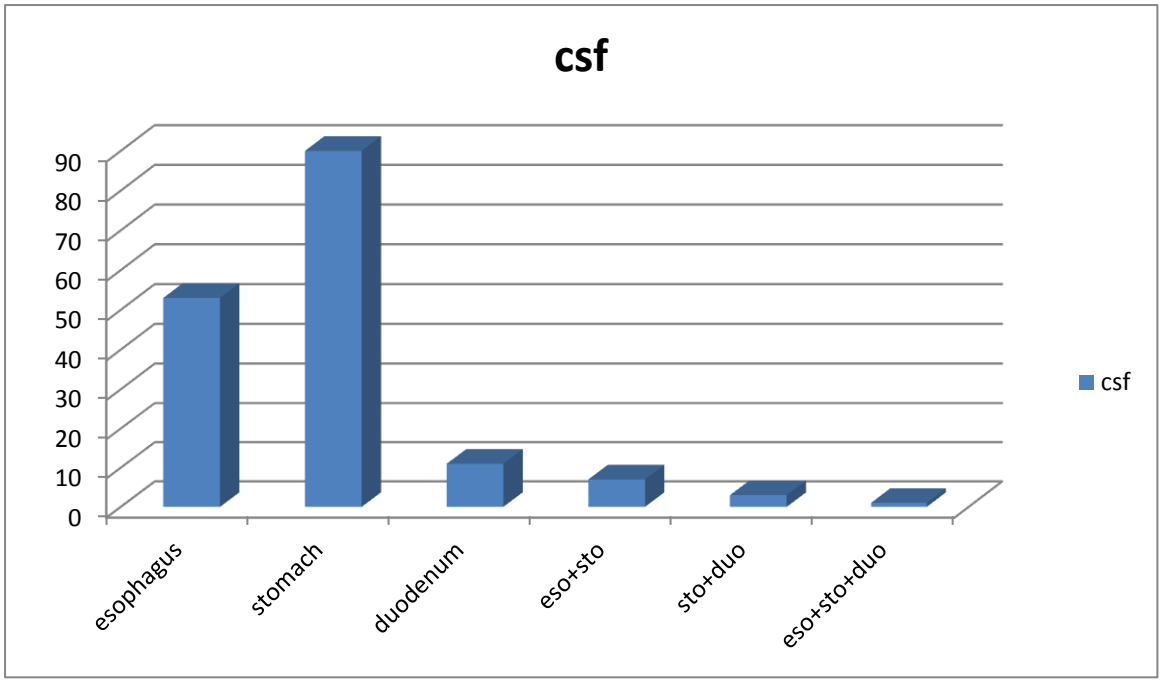


Analysis of various diseases on endoscopy showed that the most common pathology was inflammatory lesions seen in 88 (44%) of patients, of which 55(46.6%) were male patients and 33 (40.2%) were female patients, followed by Hiatus hernia and GERD were next common abnormal findings, 45 (22.5%) in thedecreasing order of the frequency of which 23 (19.5%) were males and 22 (26.8%) females. Ulcer dyspepsia was seen in 19 (6.5%) of which11 (9.3%) males and 8 (4%) females. Malignancy was common in males 9 (7.6%) patients.

Table-5 Prevalence of clinically significant endoscopic finding according to the site of lesions

s.no	CSF's	AGE 41-60	AGE 61-80	TOTAL	PERCENTAGE
1	ESOPHAGUS	39	14	53	32.1%
2	STOMACH	68	22	90	54.5%
3	DUODENUM	9	2	11	6.7%
4	ESO+STO	5	2	7	4.2%
5	STO+DUO	2	1	3	1.8%
6	ESO+STO+DUO	1	0	1	0.6%
	TOTAL	124	41	165	100%

Graph-5 Prevalence of clinically significant endoscopic findings according to the site of lesion

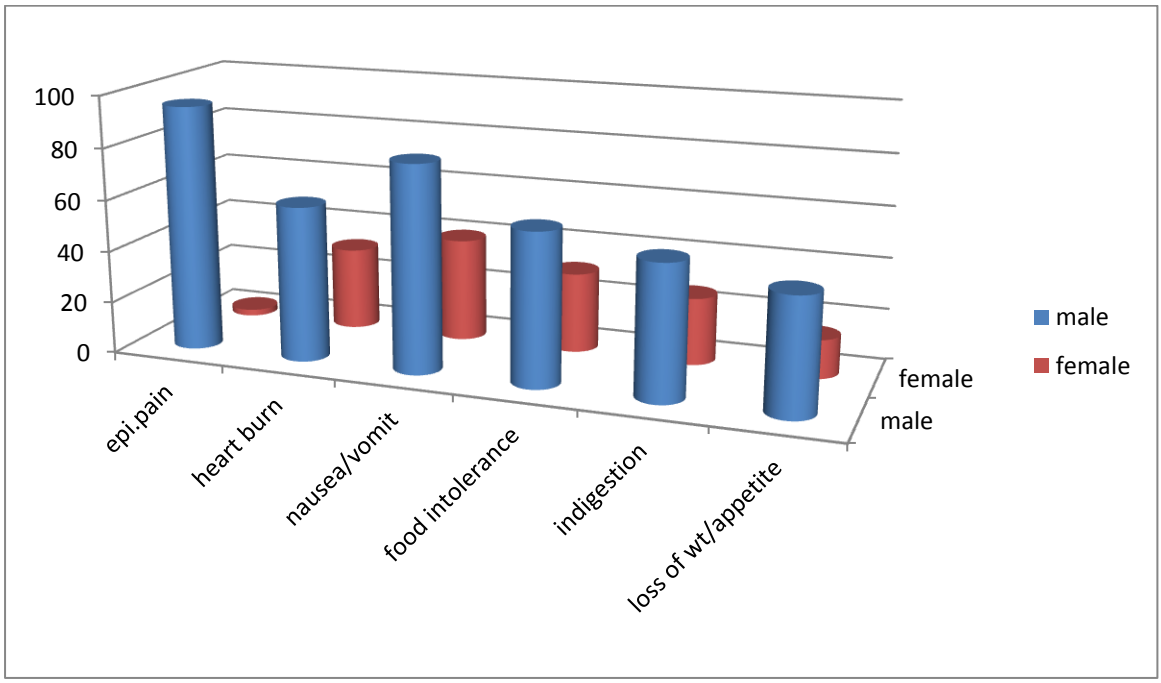


Out of 165 patients with clinically significant endoscopic findings, mostcommon pathology was seen in stomach of 90 (54.5%), patients followed by esophagus 53 (32.1%) and duodenum 11 (6.7%).

s.no	Clinical presentation	male	female	total	Percentage
1	Epigastric pain	95	50	145	72.5%
2	Heart burn	60	32	92	46%
3	Nausea/vomiting	80	40	120	60%
4	Food intolerance	59	31	90	45%
5	Indigestion	52	26	78	39%
6	Loss of weight/appetite	45	15	60	30%

Table-6 Frequency of various symptoms of dyspepsia in males and females

Graph-6 Frequency of various symptoms of dyspepsia in males and females



Out of 200 patients, the most common component of dyspepsia was epigastric pain and discomfort, seen in 145 (72.5%) patients, followed by nausea and/or vomiting 120(60%) patients, heart burn in 92 (46%) patients, food intolerance in 90 (45%) patients, indigestion in 78 (39%) patients and loss of appetite and/or weight in 60 (30%) patients.

DISCUSSION

A prospective clinico-pathological study entitled "A Clinical study of various findings in upper gastro-intestinal endoscopy in patients presenting with late onset dyspepsia at general surgery department, "RajivGandhi Govt. General Hospital" attached to Madras Medical College and Research Institute to study the endoscopic findings of dyspepsia, to detect esophagogastroduodenal carcinoma at early stages.

After informed consent 200 cases of dyspepsia were included in the study and were studied clinically as per the proforma over a period of one and half year from June 2013 to Nov 2013. All the patients underwent upper gastro-intestinal endoscopy and various findings were noted.

CLINICAL PRESENTATION:

Out of 200 patients, 145 (72.5%) patients had epigastric pain and discomfort as their chief complaint where as nausea and vomiting was present in120 (60%) patients. The other complaints were heart burn 92 (46%), food intolerance 90(45%), indigestion 78(39%) and loss of appetite and weight60(30%).

Similar study was conducted by Thomson A B R et al, in which the common presenting complaints were upper abdominal pain (34.3%), heart

burn (24.5%) and acid regurgitation (13.3%),9 the observations were comparable with that of the present study.

COMPARISON OF GENDER DISTRIBUTION

In this study 59% were male patients, 41% were female patients. The incidence of different presentations of late onset dyspepsia were common in males compared to females. Only the incidence of esophagogastritis was more in female patients.

The male / female ratio in the studies conducted by Khan N et al – 2.3:1, Ziauddin- 1.6:1, Mustapha SK et al- 1.1:1 respectively. In these studies also the majority of patients were males as observed in our study.6,40,41

In a population based study in Australia, female adults significantly out numbered males in most functional gastrointestinal disorders includes functional dyspepsia.42

COMPARISON OF VARIOUS ENDOSCOPIC FINDINGS:

In the present study, clinically significant endoscopic findings were

observed in 165 patients accounting for 82.5%. Gastritis was by far the most common finding (28%), while GERD was found in 17.5%. The next common findings were esophagitis, gastric ulcer and duodenal ulcer accounting for 12.5% each.

The percentage of cases with gastritis in this study was higher than that observed in studies by Sarwar et al and Ziauddin. The percentage of patients GERD was nearly equal to that observed by Sarwar et al.

s.no	Name of the study	Gastritis	Reflux esophagitis/ GERD
1	Sarwar et al.39	13%	20%
2	Ziauddin40	18%	14%
3	Present	28%	17.5%

Table-7

COMPARISON OF INCIDENCE OF GASTRIC MALIGNANCIES:

In this study there were 11 patients with carcimona stomach accounting for 5%, among them which 8 were male patients. Gastric malignancies were common in older age groups. Incidence of gastric malignancies observed by various authors are as follows:

s.no	NAME OF STUDY	% GASTRIC/ MALIGNANCIES
1	Choomsri P et al.5	1%
2	Khan N et al.6	3%
3	Ziauddin40	4%
4	Present study	6.5%

Table-8 The incidence of gastric malignancy in these studies is comparable with the observed in the present study.

CONCLUSION

From the present study of "A clinical study of various findings in upper gastro-intestinal endoscopy in patients presenting with late onset dyspepsia at Rajiv Gandhi Govt.General Hospitals, Madras Medical College, Chennai.

On endoscopic examination gastritis and GERD together accounted for the majority of the cases. Incidence of malignancy in the present study was observed to be 6.5% (including both gastric and esophageal malignancies).

Clinically significant endoscopic findings were observed in 82.5% ofpatients with uninvestigated dyspepsia. Most patients presented with a complex of three or more dyspeptic symptoms and the symptom profile was not predictive of the endoscopic findings.

Prevalence of large number of inflammatory lesions as a result of increased acid production, low incidence of malignancy in the study group suggests that the uninvestigated patients withdyspepsia may be initially managed medically with acid suppressive therapy.

Endoscopy may be undertaken in patients with recurrent symptoms or in whom drug therapy fails.

SUMMARY

A prospective clinico-pathological study was undertaken in RGGGH attached to Madras Medical College and Research Institute, Madras to know the various endoscopic findings in patients presenting with dyspepsia, prevalence of malignancy in these patients.

200 patients aged more than 40 years presenting with dyspepsia were evaluated.

The following were the observations:

1. Highest prevalence of late onset dyspepsia in the age group of 41-50years

2. Most common presenting complaint was epigastric pain and discomfort

3. Dyspepsia was more common in males (59%) when compared to females

4. Most common endoscopic finding was gastritis followed by GERD

5. Malignancy was diagnosed in 6.5% patients with dyspepsia.

6. Clinically significant endoscopic findings were observed in 82.5% of patientswith uninvestigated dyspepsia.

7. Most patients presented with a complex of three or more dyspeptic symptomsand the symptom profile was not predictive of the endoscopic findings. However, the high prevalence of gastritis (28%), suggests that most patients presenting with uninvestigated dyspepsia can be safely managed initially with acid suppressive drugs.

BIBLIOGRAPHY

1. Talley NJ. Dyspepsia: how to manage and how to treat?. Aliment Pharmacol Ther. 2002;16 (4) :95-104.

2. Drossman DA, Corazziari E, Talley NJ et al. The functional gastrointestinal disorders. 2nd ed. In: Diagnosis, Pathophysiology and Treatment: a Multinational Consensus. Degon: McLean, Virginia;2000.

3.Westbrook JI, McIntosh JH, Talley NJ. The impact of dyspepsia definition on prevalence estimates: considerations for future researchers. Scand J Gastroenterol. 2000; 35:227–33.

4. Talley NJ, Holtmann G. New concepts in functional gastrointestinal diseases: functional dyspepsia and its link to other disorders. In: Textbook of Gastroenterol. Philadelphia, Lippincott Williams and Wilkins;2001.

5. Choomsri P et al. Upper gastrointestinal endoscopic findings in patients presenting with dyspepsia. Thai J Surg. 2010;31:7-12.

6. Khan N et al. Upper gastrointestinal endoscopic assessment of patients presenting with dyspepsia. JPMI. 2007;21(3):212-6.

7. Thomson A B R et al. The prevalence of clinically significant endoscopic findings in primary care patients with uninvestigated dyspepsia: The Canadian Adult Dyspepsia Empiric Treatment- Prompt Endoscopy (CADET-PE) study. Aliment Pharmacol Ther. 2003; 17: 1481-91.

8. Delaney BC, Wilson S, Roalfe A et al. Cost effectiveness of initial endoscopy for dyspepsia in patients over age 50 years: A randomized controlled trial in primary care. Lancet. 2000; 356:1965-9.

9. Wiklund I, Glise H, Jerndal P et al. Does endoscopy have a positive impact on quality of life in dyspepsia ? Gastrointest Endosc. 1998;47:449-54.

10. Sandler RS, et al: The burden of selected digestive diseases in the United States. Gastroenterol. 2002; 122:1500.

11. Owen DA: Gastritis and carditis. Mod Pathol. 2003;16:325.

12. National Institute for Health and Clinical Excellence: Clinical guideline Dyspepsia. London;2004:17.

13. Hungin A, Thomas P, Bramble M, et al. What happens to patients following open access gastroscopy? An outcome study from general practice, Br J Gen Pract. 1994;44:519-21.

14. Mary Maish. Esophagus.18th ed. In: Sabiston Textbook of Surgery (2)Townsend, Beauchamp eds. Philadelphia. Saunders. 2008: pp.1049-107.

15. Jeffrey H. Esophagus and Diaphragmatic Hernia, 8th ed. In: Schwartz's Principles of Surgery, U.S.A, McGraw- Hill; 2005:pp 835-931.

16. Ganong W F. Regulation of gastrointestinal function. 20th ed. In: Review of Medical Physiology,Ganong WF, ed. USA. McGraw-Hill Company;2001: pp 439.

17. Brzozowski, Tomasz MD, Konturek et al. Involvement of Endogenous Cholecystokinin and Somatostatin in gastroprotection induced by intraduodenal fat. J Clin Gastroenterol. 1998; 27(1):125-37.

18. Kenneth A. History and Development of Flexible Endoscopy. In: Mastery of Endoscopic and Laparoscopic Surgery, Eubanks, Swanstrom, Soper, eds. USA, Lippincott Williams and Wilkins;2000:pp 3-6.

19. Bruce V. Diagnostic Upper Gastrointestinal Endoscopy, Mastery of Endoscopic and Laparoscopic Surgery, Eubanks, Swanstrom, Soper, eds. USA,Lippincott Williams and Wilkins; 2000;115-22.

20. Mitsuo, Sotaro. Fibergastroscopic Examination. 1st ed. In: Fiberscopy of diseases, Tsuneoka, Kenji, eds. Tokyo ,Igaku Shoin Ltd;1973: pp

21. Talley NJ, Vakil N. "Guidelines for the management of dyspepsia". Am J Gastroenterol. 2005;100 (10):2324-37.

22. National Institute for Health and Clinical Excellence. Dyspepsia. 2004,August.

23. Talley NJ, Stanghellini V, Heading RC, Koch KL, Malagelada JR, Tytgat GN. "Functional gastroduodenal disorders". Gut. 1999; 45 (2): 1137–42.

24. Talley NJ, Phung N, Kalantar JS . ABC of the upper gastrointestingal tract: Indigestion: When is it functional?. Br Med J. 2001; 323 (7324): 1294–7.

25. Talley NJ, Weaver AL, Tesmer DL, Zinsmeister AR (1993). "Lack of discriminant value of dyspepsia subgroups in patients referred for upper

endoscopy". Gastroenterol. 105 (5): 1378–86.

26. Dyspepsia: Managing dyspepsia in adults in primary care, evidence based clinical practical guideline, 2004; North of England dyspepsia guideline development group, center for health services, Research report no. 112, University of New Castle, ISDN 0-9540161-7-3.

27. American Gastroenterology Association Technical Review on the Evaluation of Dyspepsia. Gastroenterol. 2005;129:1756-80.

28. Goh K L. "Update of management of Helicobacter pylori infection, including drug-resistant organisms." J Gastroenterol Hepatol. 2002 17(4): 482-487.

29. Sarwar M et al. Endoscopic assessment of Dyspepsia. Pak Armed Forces Med J. 2004;54: 48-50.

30. Ziauddin. Endoscopic findings in Dyspepsia a prospective study of 200 cases J Postgrad Med Inst. 2003;17 (2) :235-9.

31. Koloski NA et al. Epidemiology and health care seeking in the functional GI disorders; a population- based study. Am J Gastroenterol. 2002;97: 2290-9.

32. Wulfen Von, Heeseman J, Butzow et al. "Detection of C. pyloridis in patients with antral gastritis and peptic ulcers by culture, compliment fixation test and immunoblot." J Clin Microbiol.1986; 24: 716-9.

ANNEXURE-A

KEY TO MASTER-CHART

OP.NO - Out patient number.

Clin- Clinical..

HPE- Histopathological Examination.

EP/A- Epigastric pain/discomfort.

HB- Heart burn.

N/V- Nausea/ Vomiting.

FI- Food intolerance.

IDG- Indigestion.

L W/A- Loss of Weight/Appetite.

PA- Pallor.

ET- Epigastric tenderness.

APD- Acid peptic disease

NS- Normal Study

Es- Esophagitis.

Ep- Esophageal polyp.

HH- Hiatus hernia.

GERD- Gastro-esophageal reflux disease.

BE- Barrett’s esophagus.

CaE- Carcinoma esophagus.

Gs- Gastritis.

GU- Gastric ulcer.

CaS- Carcinoma Stomach.

Ds- Duodenitis.

DU- Duodenal ulcer.

EG- Esophagogastritis.

GD- Gastroduodenitis.

EGD-Esophagogastroduodenitis.

RE- Reflux esophagitis.

NSP- No significant pathology.

M- Male.

F- Female.

Y- Yes.

N- No.

Sl.No	Name	Age	Sex	IP No	Clinical symptoms						signs		DIAGNOSI
					EP	HB	N/V	FI	ID	LOA	PA	ET	
1	Ravi	45	M	123365	Y	Y	Y	Y	Y	N	N	N	GERD
2	PERUMAL	47	M	187892	Y	Y	Y	N	N	N	N	N	APD
3	SARATHI	53	M	123698	Y	Y	Y	N	N	Y	Y	Y	APD
4	KUMAR	58	M	103658	Y	N	Y	Y	Y	Y	Y		APD
5	SARITHA	41	F	100236	N	Y	Y	Y	N	N	N	N	APD
6	VIJAYAL	59	F	136985	N	N	N	Y	Y	Y	Y	Y	APD
7	FAZIL	65	M	136336	Y	Y	Y	Y	Y	Y	Y	Y	APD
8	VETRISELVAN	48	M	100013	Y	N	Y	Y	Y	N	N	N	GERD
9	PUSHPA	40	F	12306	Y	Y	Y	Y	Y	Y	Y	Y	APD
10	INPHARASAN	`49	M	10068	Y	N	N	N	Y	Y	Y	Y	APD
11	MUNUSAMY	68	M	10981	Y	Y	N	Y	N	N	N	N	APD
12	VISHWA	40	M	10004	Y	Y	Y	Y	Y	Y	Y	Y	APD
13	KUPPAN	58	M	90081	Y	N	N	N	Y	Y	Y	Y	GERD
14	MAYA	42	F	133698	Y	N	N	N	N	Y	Y	Y	APD
15	VAIGAISELVAN	62	M	97865	N	Y	Y	N	N	Y	Y	Y	APD
16	PAALPAANDI	76	M	17895	N	Y	N	N	Y	Y	Y	Y	GERD
17	GANGADURAI	53	M	1436	Y	Y	Y	Y	Y	Y	Y	N	APD
18	VENNILA	47	F	19874	Y	Y	Y	N	N	N	N	Y	APD
19	CHELLADURAI	70	M	99789	`Y	Y	Y	Y	N	N	N	N	APD
20	UMASELVI	66	F	17568	Y	Y	N	N	N	N	Y	Y	APD
21	VENKATESH	`44	M	1365987	N	N	Y	Y	Y	Y	Y	Y	APD
22	`VELUSAMY	55	M	123336	Y	Y	Y	Y	Y	Y	Y	Y	APD
23	BHUVANNAMAL	56	F	45688	Y	Y	Y	N	N	Y	N	N	APD
24	PRABHAKAR	52	M	8965	Y	N	Y	Y	N	N	Y	Y	APD
25	VELMURUGAN	41	M	11456	Y	Y	N	N	Y	Y	Y	Y	APD
26	`PRASANNA	46	M	8897	Y	Y	Y	N	Y	Y	Y	Y	APD
27	CHANDRASEKAR	55	M	9968	Y	N	N	Y	Y	Y	Y	N	APD
28	PANDIYARAJ	66	M	122566	N	N	Y	Y	N	N	Y	Y	APD
29	GAYATHRI	46	F	98989	Y	Y	Y	Y	Y	Y	Y	N	APD
30	MARIYAMMAL	55	F	16556	Y	Y	N	Y	N	N	N	Y	APD
31	PUSHPAVALLI	69	F	10033	Y	Y	Y	Y	N	Y	N	N	GERD
32	SUBRAMANIAN	44	M	166678	Y	N	Y	Y	Y	N	N	Y	APD
33	VADIVELU	68	M	5648	Y	Y	Y	Y	N	N	N	Y	APD
34	VIJAYAN	56	M	456321	Y	N	Y	N	N	N	N	N	APD
35	ANDAAL	66	F	85632	Y	Y	Y	Y	Y	Y	Y	Y	APD
36	RICHARDSON	44	M	122365	N	Y	Y	Y	N	N	Y	Y	GERD
37	HARIHARAN	43	M	6985	N	Y	Y	Y	N	N	N	N	APD
38	RAJESWARI	40	F	9986	Y	Y	Y	Y	Y	Y	Y	Y	APD
39	SRINIVASAN	57	M	5555	Y	N	N	N	Y	Y	N	N	APD
40	RAJENDIRAN	66	M	65987	Y	Y	Y	N	N	N	Y	Y	APD
41	KUPPAN	48	M	25464	Y	Y	Y	Y	N	N	Y	Y	APD
42	ROJA	40	F	124539	N	Y	Y	Y	Y	N	N	N	GERD

43	PACHAIAMMAL	56	F	45632	Y	Y	N	Y	Y	Y	Y	Y	APD
44	KAVITHA	41	F	444789	N	Y	Y	Y	N	Y	N	Y	APD
45	SUDHA	49	F	103698	Y	N	Y	N	Y	N	Y	N	GERD
46	KIRUBHAKARAN	56	M	3659	Y	N	N	N	Y	Y	Y	Y	APD
47	GUHAN	56	M	897456	Y	Y	Y	Y	Y	Y	N	N	APD
48	BHAKIYALAKSHMI	59	F	14586	Y	Y	Y	Y	N	Y	N	Y	GERD
49	PANNEER SELVAM	48	M	26589	Y	Y	Y	Y	Y	Y	Y	N	APD
50	FATHIMA BEGAM	56	F	100369	Y	N	N	N	N	Y	Y	Y	GERD

51	YUVARAJAN	41	M	56987	Y	Y	Y	Y	Y	N	N	N	APD
52	ABRAHAM	54	M	78965	N	Y	Y	N	Y	N	Y	N	APD
53	KALYANA BHARATHI	69	M	2365	Y	N	N	Y	Y	Y	Y	Y	APD
54	ASHOK KUMAR	56	M	444789	Y	N	Y	N	Y	N	N	N	APD
55	KULANDHAI VEL	56	M	452236	Y	Y	Y	Y	Y	Y	Y	Y	GERD
56	PURUSHOTHAMAN	44	M	789663	Y	Y	Y	Y	Y	Y	Y	Y	GERD
57	NALLAMMAL	44	F	111456	Y	Y	Y	Y	N	N	N	N	GERD
58	KANNAKI	55	F	401145	Y	N	N	Y	Y	Y	Y	Y	APD
59	MALLIKA	56	F	11145	Y	Y	N	N	N	N	Y	Y	APD
60	CHITRA	69	F	52365	Y	Y	Y	Y	Y	Y	N	Y	APD
61	FARZANA	56	F	445669	Y	Y	Y	N	Y	Y	Y	N	APD
62	ADHIL AHAMED	62	F	445670	Y	Y	N	N	N	N	Y	N	APD
63	VIDHYALAKSHMI	44	F	102365	N	N	N	N	Y	Y	Y	Y	APD
64	VARADHARAJ	55	M	3659	Y	Y	Y	Y	Y	N	Y	Y	GERD
65	RAMANUJAM	60	M	13699	Y	N	N	N	Y	N	Y	Y	APD
66	MURALIDHARAN	56	M	100365	Y	Y	N	N	N	N	N	Y	GERD
67	BAVANI	66	F	69899	Y	Y	Y	N	N	N	Y	N	APD
68	RAJKUMAR	60	M	12132	Y	Y	N	N	N	N	Y	Y	APD
69	RAJAMMAL	69	F	100036	Y	Y	Y	Y	Y	Y	Y	Y	APD
70	MURUGAMMAL	56	F	123004	Y	N	N	N	Y	Y	Y	Y	GERD
71	SARAVANA KUMAR	63	M	12445	Y	Y	Y	N	N	N	Y	Y	APD
72	THIYAGARAJAN	46	M	144566	Y	N	N	N	Y	Y	Y	Y	GERD
73	CHINNSAMY	50	M	23659	Y	Y	Y	Y	Y	Y	Y	Y	APD
74	KUIYILI	45	F	46236	Y	N	N	N	Y	Y	Y	N	GERD
75	UMESH KUMAR	66	M	1144569	Y	Y	Y	Y	N	Y	Y	N	APD

76	PERIYASAMY	44	M	1236598	Y	Y	Y	Y	Y	Y	Y	N	APD
77	ABILASH	54	M	436736	Y	N	N	N	N	Y	Y	Y	GERD
78	BHASKARAN	70	M	10236	Y	Y	Y	N	N	N	Y	Y	APD
79	ARUNA CHANDRA	56	F	17789	N	Y	N	Y	N	Y	N	Y	APD
80	MOHAMED FAZIL	43	M	1036987	Y	N	Y	N	Y	N	Y	N	APD
81	THOMAS CHERIAN	76	M	104478	Y	Y	Y	N	Y	Y	Y	N	GERD
82	SRINIVAS REDDY	46	M	11236	Y	N	N	N	Y	Y	N	N	APD
83	NIRANJANA	56	F	13695	Y	N	N	N	N	Y	Y	N	GERD
84	BALASUBRAMANIAN	69	M	45609	Y	Y	Y	Y	Y	N	N	N	APD
85	MANIVEL	65	M	56445	N	Y	N	Y	N	Y	Y	Y	GERD
86	CHEZIAN	66	M	75569	N	Y	N	Y	Y	Y	N	Y	GERD
87	ARULMOZHI	44	M	563369	Y	Y	Y	N	Y	N	Y	N	GERD
88	RANJAN KUMAR	42	M	45778	Y	N	Y	N	Y	Y	N	Y	GERD
89	BALASUNDARAM	61	M	13025	Y	Y	Y	Y	Y	Y	Y	Y	GERD
90	DEEPAVALLI	56	F	100236	Y	N	N	N	Y	Y	Y	Y	APD
91	RAMAKRISHNAN	55	M	47789	Y	Y	Y	N	Y	Y	Y	Y	GERD
92	MOULEESWARAN	55	M	13036	Y	N	Y	Y	N	N	Y	Y	GERD
93	MADHAN KUMAR	56	M	13369	Y	N	N	N	N	N	Y	Y	GERD
94	MUTHAMMA	53	F	10069	Y	N	N	N	Y	Y	Y	Y	APD
95	DEEPAMMAL	60	F	56933	Y	N	Y	Y	Y	Y	Y	Y	GERD
96	NARAYANA MURTHY	60	M	456223	Y	N	Y	Y	Y	Y	Y	N	GERD
97	IRUTHAYA SAMY	56	M	471123	Y	Y	N	N	N	Y	Y	Y	GERD
98	RAMAKRISHNAN	60	M	13369	Y	Y	Y	N	Y	Y	Y	Y	APD
100	SUBRAMANIAN	63	M	133023	Y	N	N	Y	Y	Y	Y	N	GERD

101	MARIA	45	F	1236598	Y	Y	Y	Y	Y	Y	Y	N	APD
102	MALA PAUL	43	F	436736	Y	N	N	N	N	Y	Y	Y	GERD
103	VIJAYA RAJAN	64	M	10236	Y	Y	Y	N	N	N	Y	Y	APD
104	GOVINDHARAJAN	56	M	17789	N	Y	N	Y	N	Y	N	Y	APD
105	GOVINDHASAMY	45	M	1036987	Y	N	Y	N	Y	N	Y	N	APD
106	RAJESH	47	M	104478	Y	Y	Y	N	Y	Y	Y	N	GERD
107	KARTHIK	58	M	11236	Y	N	N	N	Y	Y	N	N	APD
108	KOWSALYA	53	F	13695	Y	N	N	N	N	Y	Y	N	GERD
109	VENKATESAN	65	M	45609	Y	Y	Y	Y	Y	N	N	N	APD
110	SURESH	42	M	56445	N	Y	N	Y	N	Y	Y	Y	GERD
111	LOGU	75	M	75569	N	Y	N	Y	Y	Y	N	Y	GERD
112	JAYARANI	56	F	563369	Y	Y	Y	N	Y	N	Y	N	GERD
113	NAGAPPAN	54	M	45778	Y	N	Y	N	Y	Y	N	Y	GERD
114	ANBU	56	M	13025	Y	Y	Y	Y	Y	Y	Y	Y	GERD
115	KASTHURI	54	F	100236	Y	N	N	N	Y	Y	Y	Y	APD
116	NISHANTHI	46	F	47789	Y	Y	Y	N	Y	Y	Y	Y	GERD

117	SENTHILKUMAR	44	M	13036	Y	N	Y	Y	N	N	Y	Y	GERD
118	GUNASEKAR	44	M	13369	Y	N	N	N	N	N	Y	Y	GERD
119	IYAPPAN	46	M	10069	Y	N	N	N	Y	Y	Y	Y	APD
120	MAGESWARI	47	F	56933	Y	N	Y	Y	Y	Y	Y	Y	GERD
121	PRABHAKAR	42	M	456223	Y	N	Y	Y	Y	Y	Y	N	GERD
122	RAJALAKSHMI	41	F	471123	Y	Y	N	N	N	Y	Y	Y	GERD
123	JANAKIRAMAN	40	M	13369	Y	Y	Y	N	Y	Y	Y	Y	APD
124	MOHAMED RIAZ	41	M	133023	Y	N	N	Y	Y	Y	Y	N	GERD
125	VETRISELVI	48	F	1236598	Y	Y	Y	Y	Y	Y	Y	N	APD

1

126	GUNASEKARAN	45	M	1236598	Y	Y	Y	Y	Y	Y	Y	N	APD
127	HARINATH	43	M	436736	Y	N	N	N	N	Y	Y	Y	GERD
128	PONGANESH	64	M	10236	Y	Y	Y	N	N	N	Y	Y	APD
129	SHANMUGAM	56	M	17789	N	Y	N	Y	N	Y	N	Y	APD
130	KARTHIKA	45	F	1036987	Y	N	Y	N	Y	N	Y	N	APD
131	MANIKANDAN	47	M	104478	Y	Y	Y	N	Y	Y	Y	N	GERD
132	MANIMARAN	58	M	11236	Y	N	N	N	Y	Y	N	N	APD
133	LALITH	53	M	13695	Y	N	N	N	N	Y	Y	N	GERD
134	ASHOK	65	M	45609	Y	Y	Y	Y	Y	N	N	N	APD
135	VISWAKANTH	42	M	56445	N	Y	N	Y	N	Y	Y	Y	GERD
136	REKHA	75	F	75569	N	Y	N	Y	Y	Y	N	Y	GERD
137	KAMATCHI	56	F	563369	Y	Y	Y	N	Y	N	Y	N	GERD
138	VEERAMANI	54	M	45778	Y	N	Y	N	Y	Y	N	Y	GERD
139	GAYATHRI	56	F	13025	Y	Y	Y	Y	Y	Y	Y	Y	GERD
140	RAJKUMAR	54	M	100236	Y	N	N	N	Y	Y	Y	Y	APD
141	MUTHUKRISHNAN	46	M	47789	Y	Y	Y	N	Y	Y	Y	Y	GERD
142	VIGNESH	44	M	13036	Y	N	Y	Y	N	N	Y	Y	GERD
143	NAGALAKSHMI	44	F	13369	Y	N	N	N	N	N	Y	Y	GERD
144	SRIDHAR	46	M	10069	Y	N	N	N	Y	Y	Y	Y	APD
145	PRAKASH	47	M	56933	Y	N	Y	Y	Y	Y	Y	Y	GERD
146	RAVI	42	M	456223	Y	N	Y	Y	Y	Y	Y	N	GERD
147	UMAMAHESWARI	41	F	471123	Y	Y	N	N	N	Y	Y	Y	GERD
148	JAYAKODI	40	F	56445	N	Y	N	Y	N	Y	Y	Y	GERD
149	MOHAN	41	M	75569	N	Y	N	Y	Y	Y	N	Y	GERD
150	RADHA	49	F	563369	Y	Y	Y	N	Y	N	Y	N	GERD

151	VIGNESH	57	M	1236598	Y	Y	Y	Y	Y	Y	Y	N	APD
152	JOSEPH	45	M	436736	Y	N	N	N	N	Y	Y	Y	GERD
153	PRADEEP KUMAR	52	M	10236	Y	Y	Y	N	N	N	Y	Y	APD
154	PRATHEEBA	53	F	17789	N	Y	N	Y	N	Y	N	Y	APD
155	UMA	56	F	1036987	Y	N	Y	N	Y	N	Y	N	APD
156	UMA	45	F	104478	Y	Y	Y	N	Y	Y	Y	N	GERD
157	KARTHIKEYAN	67	M	11236	Y	N	N	N	Y	Y	N	N	APD
158	BHOJAN	42	M	13695	Y	N	N	N	N	Y	Y	N	GERD
159	THANGARAJAN	55	M	45609	Y	Y	Y	Y	Y	N	N	N	APD
160	SUGANYA	45	F	56445	N	Y	N	Y	N	Y	Y	Y	GERD
161	KEERTHANA	42	F	75569	N	Y	N	Y	Y	Y	N	Y	GERD
162	PRIYA	46	F	563369	Y	Y	Y	N	Y	N	Y	N	GERD
163	SIVA	71	M	45778	Y	N	Y	N	Y	Y	N	Y	GERD
164	GANGADHARAN	47	M	13025	Y	Y	Y	Y	Y	Y	Y	Y	GERD
165	KALPANA	49	F	100236	Y	N	N	N	Y	Y	Y	Y	APD
166	ABIRAMI	66	F	47789	Y	Y	Y	N	Y	Y	Y	Y	GERD
167	SEKAR	60	M	13036	Y	N	Y	Y	N	N	Y	Y	GERD
168	VANATHI	47	F	13369	Y	N	N	N	N	N	Y	Y	GERD
169	NIBURAJAN	56	F	10069	Y	N	N	N	Y	Y	Y	Y	APD
170	VIGNESH	41	M	56933	Y	N	Y	Y	Y	Y	Y	Y	GERD
171	MALLIKA	54	F	456223	Y	N	Y	Y	Y	Y	Y	N	GERD
172	KALPANA	42	F	471123	Y	Y	N	N	N	Y	Y	Y	GERD
173	ANANDHAVALLI	46	F	13036	Y	N	Y	Y	N	N	Y	Y	GERD
174	SENTHAMARAI	54	F	13369	Y	N	N	N	N	N	Y	Y	GERD
175	AMEERJAAN	64	F	10069	Y	N	N	N	Y	Y	Y	Y	APD

176	MANIMEGALAI	78	F	1236598	Y	Y	Y	Y	Y	Y	Y	N	APD
177	KARPAGAM	64	F	436736	Y	N	N	N	N	Y	Y	Y	GERD
178	SABEER	45	F	10236	Y	Y	Y	N	N	N	Y	Y	APD
179	INDHUMATHI	58	F	17789	N	Y	N	Y	N	Y	N	Y	APD
180	DHANDAPANI	68	M	1036987	Y	N	Y	N	Y	N	Y	N	APD
181	ARJUN	48	M	104478	Y	Y	Y	N	Y	Y	Y	N	GERD
182	LEENA ROSELIN	66	F	11236	Y	N	N	N	Y	Y	N	N	APD
183	PALANI	53	M	13695	Y	N	N	N	N	Y	Y	N	GERD
184	GOPUKUMAR	49	M	45609	Y	Y	Y	Y	Y	N	N	N	APD
185	RAJI	61	F	56445	N	Y	N	Y	N	Y	Y	Y	GERD
186	KALAIVANI	55	F	75569	N	Y	N	Y	Y	Y	N	Y	GERD

187	MAHABOOBEE	61	F	563369	Y	Y	Y	N	Y	N	Y	N	GERD
188	THULASI	41	M	45778	Y	N	Y	N	Y	Y	N	Y	GERD
189	PANDIYARAJ	58	M	13025	Y	Y	Y	Y	Y	Y	Y	Y	GERD
190	MELVIN	54	M	100236	Y	N	N	N	Y	Y	Y	Y	APD
191	PRIYA	45	F	47789	Y	Y	Y	N	Y	Y	Y	Y	GERD
192	CHRIS ALEN PRINCY	43	M	13036	Y	N	Y	Y	N	N	Y	Y	GERD
193	SHANTHI	45	F	13369	Y	N	N	N	N	N	Y	Y	GERD
194	HEMAMALINI	55	F	10069	Y	N	N	N	Y	Y	Y	Y	APD
195	NANDAKUMAR	65	M	56933	Y	N	Y	Y	Y	Y	Y	Y	GERD
196	BHUVANESHWARI	62	M	456223	Y	N	Y	Y	Y	Y	Y	N	GERD
197	NANDHINI	49	F	471123	Y	Y	N	N	N	Y	Y	Y	GERD
198	MANIMEGALAI	60	F	13369	Y	Y	Y	N	Y	Y	Y	Y	APD
199	KARPAGAM	58	F	133023	Y	N	N	Y	Y	Y	Y	N	GERD
200	SABEER	70	F	1236598	Y	Y	Y	Y	Y	Y	Y	N	APD

ANNEXURE-C

CLINICAL PROFORMA

PATIENT DETAILS:

ON ADMISSION:

Name :

Age :

Sex :

IP No. :

Duration of symptoms :

Co-morbid illness :

DM : yes / no IHD / CAD : yes / no

HT : yes / no

CLINICAL EXAMINATION:

Pulse :

BP :

Anemia : yes / no

Icterus : yes / no

Pedal edema : yes / no

CVS:

RS:

P/A:

INVESTIGATIONS & INTERVENTION DONE: -

USG ABD:

CECT ABD:

OGD SCOPY:

BIOPSY TAKEN: (YES/NO)(REPORT IF YES)